Vol. 66, No. 6 November, 1959

Psychological Review

RICHARD L. SOLOMON, Editor
Harvard University

CONTENTS

A Possible Artifact in Electroencephalography.....JOHN L. KENNEDY 347

A Model of Hypothesis Behavior in Discrimination Learning Set.....MARVIN LEVINE 353

Activation: A Neuropsychological Dimension.....ROBERT B. MALMO 367

The Relational Determination of Perceived Size.....IRVIN ROCK AND SHELDON EBENHOLTZ 387

This is the last issue of Volume 66. Title page and index for the volume appear herein.

PUBLISHED BIMONTHLY BY THE
AMERICAN PSYCHOLOGICAL ASSOCIATION, INC.

Editorial Consultants

Joe K. Adams
Gordon W. Allport
Elliot Aronson
Solomon E. Asch
John W. Atkinson
Robert F. Bales
Gustav Bergmann
Edgar F. Borgatta
Roger W. Brown
B. R. Bugelski
C. J. Burke
Robert R. Bush
John B. Carroll
Dorwin Cartwright
Russell M. Church
Lee J. Cronbach
James Deese
Carl P. Duncan
Ward Edwards
Charles W. Eriksen
Leon Festinger
Wendell R. Garner
Henry Gleitman
Clarence H. Graham
Bert F. Green
Eric G. Heinemann
D. O. Hebb
Harry Helson
R. J. Herrnstein
Francis W. Irwin
Harold H. Kelley
E. Lowell Kelly
Herbert C. Kelman
R. Duncan Luce
Richard D. Mann
Donald R. Meyer
Charles E. Osgood
Harold Schlosberg

(A large group of psychologists has kindly agreed to assist in the editing of manuscripts submitted to the *Psychological Review*. In each issue the Editor will list in the *Review* those editorial consultants who have helped him process manuscripts for that issue, so that their efforts may be gratefully acknowledged.)

The *Psychological Review* is devoted to articles of theoretical significance to any area of scientific endeavor in psychology. Except for occasional articles solicited by the Editor, manuscripts exceeding 7,500 words will not be accepted. Ordinarily manuscripts which consist primarily of original reports of research should be submitted to other journals. However, reports of original research may be included in a major theoretical article which attempts to integrate several related original studies.

Because of the large number of manuscripts submitted, there is an inevitable publication lag of several months. Authors may avoid this delay if they are prepared to pay the costs of publishing their own articles; the appearance of articles by other contributors is not thereby delayed.

Tables, footnotes, and references should appear on separate pages; all of these, as well as the text, should be typed double-spaced throughout, in all manuscripts submitted.

All manuscripts must be submitted in duplicate.

Original figures are prepared for publication; duplicate figures may be photographic or pencil-drawn copies. Authors should retain a copy of the manuscript, in case there is loss in the mail. Manuscripts should be addressed to the Editor, Richard L. Solomon, Emerson Hall, Harvard University, Cambridge 38, Mass.

Arthur C. Hoffman
Managing Editor

Helen Orr
Production Manager

Sarah Womack
Editorial Assistant

Published Bimonthly by the
AMERICAN PSYCHOLOGICAL ASSOCIATION, INC.
Prince and Lemon Streets, Lancaster, Pa.
1333 Sixteenth St. N. W., Washington 6, D. C.

$8.00 volume $1.50 issue

Second-class postage paid at Lancaster, Pa.

Send all communications, including address changes, to 1333 Sixteenth St. N.W., Washington 6, D. C. Address changes must arrive by the 10th of the month to take effect the following month. Undelivered copies resulting from address changes will not be replaced; subscribers should notify the post office that they will guarantee second-class forwarding postage. Other claims for undelivered copies must be made within four months of publication.

© 1959 by the American Psychological Association, Inc.

VOL. 66, NO. 6 NOVEMBER, 1959

THE PSYCHOLOGICAL REVIEW

A POSSIBLE ARTIFACT IN ELECTROENCEPHALOGRAPHY [1]

JOHN L. KENNEDY
Princeton University

Although it is now well established that the brain does produce a great variety of electrical patterns, the *oscillating potentials*, such as the alpha rhythm, have been given particular prominence as examples of some special kind of electrical activity of neurons. The proposal has been entertained, for example, that brain waves may be produced by summation of action potential spikes. In 1948, Jasper suggested that these "waves" indicate spontaneous rhythmic fluctuations in the local excitability of groups of neurons in the cerebral cortex. He further suspected that there may be certain specialized cells in the cortex that produce these rhythmic oscillations in potential. In a later paper (Li, McLennan, & Jasper, 1952) it was clearly demonstrated by simultaneous recording that the action potential spikes of neuronal activity and the slower oscillations of "brain waves" were quite uncorrelated. The authors conclude, "Brain waves do seem to be phenomena of a different order. The most plausible hypothesis is that they represent synchronized oscillations in membrane potentials, possibly involving small interneurons and dendrites in the cortical matrix, oscillations which would have a definite effect upon neuronal excitability, but not dependent upon neuronal discharge" (p. 657).

Lindsley (1952) has proposed that "alpha activity" of brain cells may go on without exhibiting a recordable alpha rhythm. He suggests that it is only when thousands of cells are responding in a synchronized manner that a recordable alpha rhythm appears. The process of synchronization or summation appears to be at the heart of theoretical speculation about the source and meaning of the normal EEG. It is the purpose of this paper to propose a model by means of which some aspects of synchronization may be understood.

Since the publication of a paper on the anterior temporal rhythm (Kennedy, Gottsdanker, Armington, & Gray, 1948) the author has been attempting to understand the nature of the normal electroencephalogram, in particular the mechanism for synchronizing electrical activity of the brain which would account for the low frequency oscillations so commonly observed. In the course of these investigations, the possibility

[1] The author wishes to thank the Carnegie Corporation of New York, Tufts University, and the RAND Corporation for financial support. Many unpublished investigations by Robert M. Gottsdanker, John C. Armington, Florence E. Gray, William H. Emmons, and Robert Hennessey have contributed substantially to the hypothesis and supporting evidence.

of an "artifact," produced by the physical structure and suspension of the brain, has appeared as an hypothesis to account for at least some of the oscillating potentials recorded from the scalp. In brief, the hypothesis requires three statements about the living brain to be true. They are: (*a*) the living brain is a gel; (*b*) the living brain is at a different potential than skull and scalp and different parts are at different potentials with respect to each other; (*c*) the brain is mechanically pulsed. With the publication in 1955 of Bering's "Choroid Plexus and Arterial Pulsation of Cerebrospinal Fluid—Demonstration of the Choroid Plexuses as a Cerebrospinal Fluid Pump," the last link in the chain of evidence has been made available. The hypothesis that emerges is at least simple and should be testable in a variety of ways.

THE MODEL

The living brain may be described, from a gross structural point of view, as an encapsulated, hollow gel mass, supported by cerebrospinal fluid both internally and externally. Because it exists in the solid box of the skull and is pentrated by large and small blood vessels in great abundance, the internal pressure varies with the amount of fluid (blood, cerebrospinal fluid), the existence of damping mechanisms (such as the fontanel) and probably other physical controlling mechanisms.

One may suppose that this gel mass, because of the continuous chemical activity of oxidation-reduction, is at a different potential than the rather inert skull and skin of the head. This seems to be a resonable assumption and is supported by Libet and Gerard's (1941) demonstration of anterior-posterior DC potential differences in the frog brain of as much as .5–2mv.

We know that a charged body, moving in relation to fixed electrodes, will produce a signal proportional to the magnitude of the potential difference between electrodes and source and the amplitude and direction of the motion. The use of the corneoretinal potential difference by Carmichael and Dearborn (1947) in recording the movements of the eyes in reading is an example of this kind of mechanism.

The living brain may be described as a gel. In searching for a common substance of about the same coefficient of elasticity, our best approximation has been ordinary commercial gelatin. If one sets such a gel in an unglazed, ceramic vessel of about the thickness of the skull, a model of the gross physical properties of the brain and skull is obtained. Standard EEG electrodes may be fixed to the exterior surface of the vessel as shown in Fig. 1.

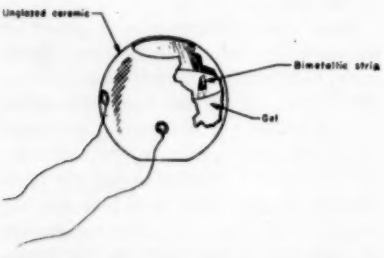

FIG. 1. Simple model of brain and skull.

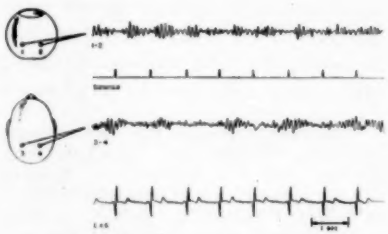

FIG. 2. Output of model compared with alpha rhythm using the repetition rate of the *S*'s EKG to drive the model.

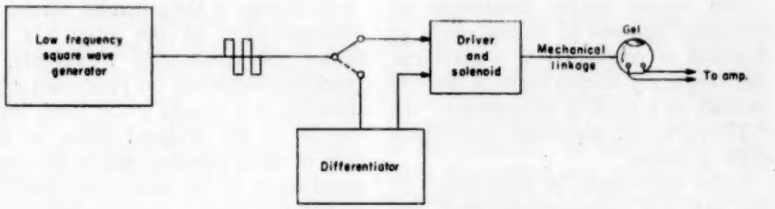

FIG. 3. System for mechanical driving of gel.

We produce a chemical reaction (with an accompanying electrical field) in the gel by plunging a bimetallic couple into it, as shown in Fig. 1. If this model is mechanically pulsed, trains of oscillations with a frequency of around 10 cycles/sec. are recorded with standard EEG instrumentation. Figure 2 shows the output of the model compared with occipital alpha rhythm simultaneously recorded. The mechanical drive for the model, in this case, was obtained by using the *S*'s own EKG to pulse the ceramic vessel, as illustrated in Fig. 3.

Evidence of a source of internal mechanical pulsing of the brain gel is supplied by Bering's investigation of the transfer of mechanical energy from the heart beat to cerebrospinal fluid via the choroid plexuses. It seems reasonable to suppose that the pumping action of the choroid plexuses may mechanically drive the surrounding brain tissue, particularly tissue that is relatively free to oscillate. The position, shape, and suspension of the occipital lobes makes them good candidates for the role of mechanical oscillators at the back of the head. The temporal lobes similarly would appear to provide a second source of oscillation in the temporal region. It might be possible to account for the localization of the "alpha" rhythm and the anterior temporal rhythm (Kennedy et al., 1948) by reference to the position of these lobes of the brain.

The frequency of the electrical signals of mechanical oscillation in a gel is apparently a function of many variables. The most important determiner seems to be the coefficient of elasticity of the gel. In our models, the tuned frequency varies with the "tightness" of the gel. As the gel melts after removal from refrigeration, the tuned frequency of oscillation decreases from frequencies of 10–12 cycles/sec. to 6–8 cycles/sec. as the gel becomes more liquid. Figure 4 presents a typical frequency amplitude graph obtained from a gel model pulsed by the apparatus shown in Fig. 3. As the repetition rate is increased from 8 to 20 constant-amplitude mechanical pulses/sec., the response of the gel exhibits a rather sharp maximum at between 9 and 10 cycles/sec., with a second maximum around 18 cycles/sec., the first harmonic. The similarity of this plot to a frequency analysis of the EEG is quite striking.

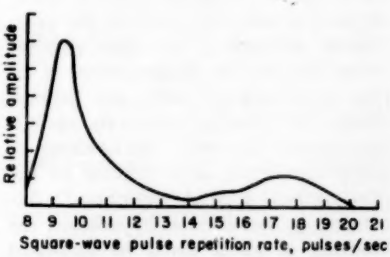

FIG. 4. Resonance of the gel model.

The second major determiner of frequency appears to be the mode or direction of oscillation of a gel mass. An irregularly shaped mass of gel will oscillate in a band of frequencies from 9–12 cycles/sec., dependent upon which mode is active.

Local differences in consistency of the gel will produce different frequencies. We have "localized" a round gum eraser embedded on the surface in a brain-sized gel mass by the "slow waves" produced in the near vicinity of the eraser. Apparently an object of different consistency than the gel will affect the mechanical properties in such a way as to produce a different local frequency of oscillation.

Another important set of variables in the control of frequency appears to be the force pattern and repetition rate of the mechanical drive. By varying the frequency and direction of the mechanical pulse one may produce a variety of frequencies of the gel. One of the peculiar characteristics of the normal EEG is "spindling," a periodic waxing and waning of amplitude. Although this appears to be a "beat" phenomenon, we have been able to produce beautiful, regular 10 cycle/sec. spindling by pulsing a gel model with a square-wave pulse at a repetition rate of 1.5 pulses/sec.

TUNING AND DETUNING OF THE LOBES

One of the controlling factors for the normal EEG is the effect of opening and closing the eyes on the presence or absence of the alpha rhythm. Since blocking or disappearance of alpha is associated with any sudden change in stimulus conditions, including opening the eyes, the blocking of alpha has often been related to the psychological state of "attention." According to the present hypothesis, the blocking of alpha could be produced by detuning the mechanical oscillator or by change in the potential difference between parts of the brain. If we follow the mechanical hypothesis, a possible mechanism for producing such detuning is a change in the local blood supply to the occipital lobes, in the case of alpha rhythm, and to the temporal lobes, in the case of the anterior temporal rhythm. Darrow and Pathman (1943) have shown that cerebral constriction typically occurs in conditions favoring increased alpha potential. Darrow, McCulloch, Green, Davis and Garol (1944) have also reported alternate blanching and reddening of the surface of the cat cortex with stimulation of the autonomic nervous system. One may suppose that "attending" results in engorgement of the brain with blood to detune the gel oscillators. In fact, Shepard (1906) has demonstrated on trephined *S*s an immediate increase in brain volume following sudden stimulation. Shepard also observed an increase of brain volume when the *S* went to sleep, a fact that correlates well with the disappearance of alpha during sleep.

A "CRUCIAL" EXPERIMENT

The present hypothesis about the production of the oscillating potentials of the normal EEG, although simple, proposes that the normal EEG results from the interaction of many variables. Hence, a crucial experiment is somewhat difficult to design. Direct measurement of mechanical brain oscillation appears full of special measurement difficulties. Shepard's (1906) experiments with trephined *S*s have suggested a way of testing the hypothesis without interfering with the system by measurement procedures.

Because of failure to record the anterior temporal rhythm (Kennedy et al., 1948) from more than 25 to 30% of *S*s, we became interested in persons who, under normal conditions, do not

have typical alpha rhythm either. Possibly 5% of a group of normal *S*s will have very poor alpha rhythm, even on repeated testing and a few show no alpha or anterior temporal rhythm at all. They seem to be perfectly normal people, but no brain waves!

An adult male *S* with a skull opening, covere by skin, over the left ear was found. The opening is roughly circular, about an inch in diameter. The injury was received in an automobile accident in his early youth. The opening is large enough to exhibit a visible pulse in the skin covering. Many EEG records were taken on this *S* with the conclusion that he is one of the relatively rare people who do not exhibit the alpha rhythm. The experiment, then, was to determine, by restoring the intracranial pressure and pulse to their normal condition, whether or not alpha would appear. Bering (1955) states, "The damping effect of the open fontanel is well demonstrated by the change in the shape of the wave [pluse wave] after the closure of the fontanel. In infants the pulse wave is rounded and without the additional peaks in the descending phase. When the fontanel closes and the skull becomes a relatively more solid box, the pulse wave becomes more sharply peaked and the peaks of the descending phase become prominent" (p. 169).

A flat, rigid plastic plate was shaped to fit into the skull defect over the skin of the *S* described above. The plate was held in place under mild pressure with a head band so that the visible pulse just disappeared. Under these conditions, the *S* began to exhibit large bursts of normal 11 cycle/sec. alpha soon after closing his eyes. Eyes

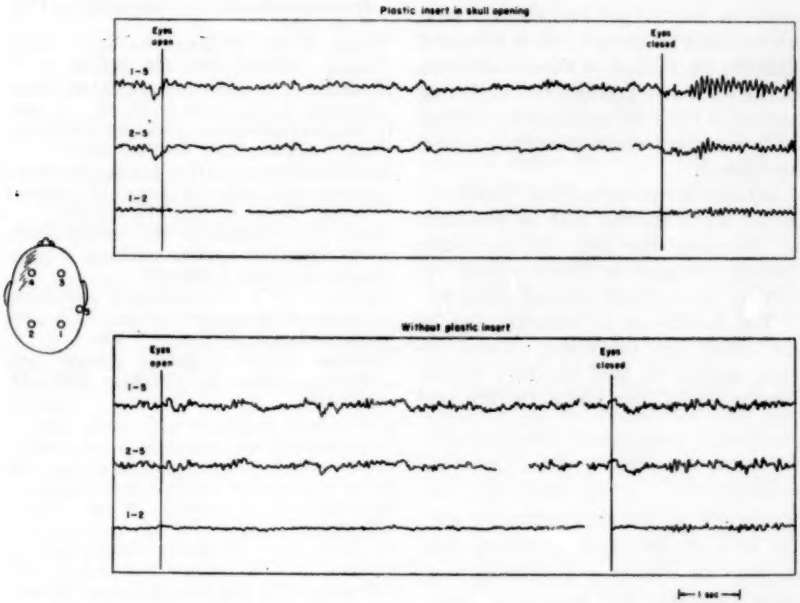

FIG. 5. Discovery of alpha rhythm in a nonalpha *S* by removing the effects of a damping mechanism.

open blocked the alpha, eyes closed produced it. No change in subjective state was reported by the *S* during the times alpha was present. Tests made with the plastic plate and head band removed before and after the critical tests were negative for the presence of alpha in any similar amount. Figure 5 illustrates the difference in an eyes open–eyes closed cycle with the plastic plate in place and removed.

SUMMARY

A simple electromechanical theory for the production of the normal electroencephalogram has been outlined. The theory requires three sets of conditions in the brain: (*a*) an electrically-charged state, presumed to arise through chemical oxidation reduction reactions, (*b*) a gel mass, with a coefficient of elasticity approximated by commercial gelatin, and (*c*) a periodic mechanical pulse, to set the gel into oscillation at its resonant frequency. It is proposed that the interaction of these conditions in the skull may produce the oscillating potentials recorded from normal human *S*s by standard electroencephalographic equipment.

It is further proposed that "blocking" of the alpha rhythm may be produced by detuning the lobes of the brain through changes in blood supply via cortical vasoconstriction and dilation.

The results of a "crucial" test of the theory are described. These results appear to lend credence to the interpretation that alpha rhythm and the anterior temporal rhythm may arise from mechanical oscillation of the gel of the living brain, not necessarily from synchronization of neural activity directly.

REFERENCES

BERING, E. S. Choroid, plexus and arterial pulsation of cerebrospinal fluid—Demonstration of the choroid plexuses as a cerebrospinal fluid pump. *Arch. Neurol. Psychiat.*, 1955, **73**, 165–172.

CARMICHAEL, L., & DEARBORN, W. F. *Reading and visual fatigue.* Boston: Houghton Mifflin, 1947.

DARROW, C. W., & PATHMAN, J. H. The role of blood pressure in electroencephalographic changes during overventilation. *Fed. Proc.*, 1943, **2**, 9.

DARROW, C. W., MCCULLOCH, W. S., GREEN, J. R., DAVIS, E. W., & GAROL, H. W. Parasympathetic regulation of high potentials in the electroencephalogram. *Arch. Neurol. Psychiat.*, 1944, **52**, 337–8.

DARROW, C. W., GREEN, J. R., DAVIS, E. W., & GAROL, H. W. Parasympathetic regulation of high potentials in the electroencephalogram. *J. Neurophysiol.*, 1944, **7**, 217–226.

JASPER, H. H. Charting the sea of brain waves. *Science*, 1948, **108**, 343–347.

KENNEDY, J. L., GOTTSDANKER, R. M., ARMINGTON, J. C., & GRAY, F. E. A new electroencephalogram associated with thinking. *Science*, 1948, **108**, 527–529.

LI, C., MCLENNAN, H., & JASPER, H. Brain waves and unit discharge in cerebral cortex. *Science*, 1952, **116**, 656–657.

LIBET, B., & GERARD, R. W. Steady potential fields and neurone activity. *J. Neurophysiol.*, 1941, **4**, 438–455.

LINDSLEY, D. B. Psychological phenomena and the electroencephalogram. *EEG Clin. Neurophysiol.*, 1952, **4**, 443–456.

SHEPARD, J. F. Organic changes and feeling. *Amer. J. Psychol.*, 1906, **17**, 522–584.

Psychological Review
Vol. 66, No. 6, 1959

A MODEL OF HYPOTHESIS BEHAVIOR IN DISCRIMINATION LEARNING SET [1]

MARVIN LEVINE [2]

University of Wisconsin

In discrimination learning, or learning-set experiments, one typically measures the percentage of correct responses on a given trial (or block of trials, or, in learning set, on a block of problems) and ignores the changes in other systematic response patterns. An alternative approach was taken by Krechevsky (1932) who first measured position preferences, position alternation, and light-going tendencies in the white rat in a discrimination problem. He described each tendency as a "hypothesis" (H), which may be defined as a specifiable pattern of response to a selected stimulus set. Harlow (1950) demonstrated other Hs by the monkey in the discrimination learning-set experiment, and Goodnow and several co-workers (Goodnow & Pettigrew, 1955; Goodnow & Postman, 1955; Goodnow, Shanks, Rubinstein, and Lubin, 1957) demonstrated patterns of response in humans.

While these researchers have found that such analyses provide useful insights into the nature of the learning process, the methods of measurement were selected for the specific demonstration at hand. No comprehensive picture of H behavior was available to suggest the total set of Hs which existed, to insure a lack of confounding in the measurement of various Hs, and to show the proportion of behavior under the control of each H. The purpose of this paper is to provide such a picture, to describe a mathematical model of H behavior which permits an analysis having the following characteristics: (*a*) The operation of a large number of Hs can be analyzed simultaneously. As many as nine will be so analyzed, although this number is by no means an upper limit. (*b*) The relative strength of each H, i.e., the proportion of responses controlled by each H, can be demonstrated at successive stages of the experiment. (*c*) The measure of a given H is uninfluenced by the presence of other Hs. (*d*) The analysis is independent of the particular reward and stimulus sequences employed. For example, the correct stimulus may be permitted to perseverate on one side for three consecutive trials, or not, or may alternate from side to side as frequently as it perseverates, or may not, etc.

This model will be applied to the behavior of the rhesus monkey in a discrimination learning-set experiment in which each problem is presented for only three trials. The stimuli in this experiment consist of two objects per problem, response to one object producing a reward (a raisin or peanut), response to the other going unrewarded. Every three trials the

[1] This work was supported in part by Grant M-772, National Institutes of Health, and in part by funds received from the Graduate School of the University of Wisconsin.

This paper is a shortened version of a dissertation submitted to the Department of Psychology at the University of Wisconsin in partial fulfillment of the requirements for the Ph.D. degree. Copies of the expanded version may be had upon request. The author wishes to thank H. F. Harlow, L. B. Wyckoff, R. E. Bowman, and N. L. Guttman for the encouragement and stimulation they provided.

[2] Now at Indiana University.

problem is changed by introducing two new objects, and several hundred problems are typically presented in this manner. A more detailed description of the learning-set procedure is given by Harlow (1949).

No analysis will be made in this paper of data beyond the third trial of any problem. The analysis may be applied to learning-set experiments utilizing longer problems simply by ignoring all trials past the third trial. The description of the behavior in such cases applies only to the first-three-trial sets. Attempts are currently being made to generalize the model in a more satisfactory way. Work is now in progress, for example, extending the model to oddity learning-set data in which each problem is presented for 12 trials and to single discrimination problems several hundred trials in length.

ASSUMPTIONS AND DEFINITIONS

The first assumption is that if an H is appearing, its occurrence will be manifested over all three trials of a given problem. Suppose, for example, that an *S* has a position preference on one problem. The only acceptable manifestation of this preference will be response to one position for the three trials. Response to this position for two out of the three trials will not serve as an instance of this H.

The second assumption is that the stimuli determining a response go no further back than the immediately preceding trial. A double-alternation H, for example, will not be considered, since the response on Trial n requires residual stimulation from the response on Trial n-2.

Given these two restrictions, the definition of a few symbols will provide sufficient background for describing the set of Hs which are considered in this paper. In any learning-set experiment the *E* decides in advance the sequence of positions which the reward (and rewarded object) will take. The reward sequence may go *LLR* on the first problem, *RRR* on the second problem, etc. He records the outcome for the three trials as $++-$, $-+-$, etc., and from the combination of *L*, *R*, and $+$, $-$ sequences can deduce the *S*'s responses. There are then three sets of symbols that are necessary: The reward sequences, the response sequences, and the outcome sequences. For any three-trial problem there are eight possible reward sequences. These may be paired into symmetrical pairs of (*LLL*, *RRR*), (*LLR*, *RRL*), (*LRL*, *RLR*), and (*LRR*, *RLL*). From the standpoint of the subsequent analysis the pairs may be combined. That is, the important property is how the reward varies rather than the particular side on which it appears on a given trial. If the sequence goes *LLL*, the important fact is that the food was on one side for three trials. The additional fact that it was the left side is here irrelevant. This consideration reduces the reward sequences to four: *AAA*, *AAB*, *ABA*, and *ABB*, where *A* is defined as the location of the reward on the first trial, and *B* is defined as the other position. A similar combining applies to the *S*'s responses. The four resulting response sequences will be symbolized by *III*, *IIO*, *IOI*, *IOO*, where *I* is the position responded to on the first trial, and *O* is the other position. Whereas the reward and response sequences reduce to four, the $+$, $-$ outcome sequences will not be combined so that all eight of these will be used.

The manifestation of the Hs considered in this paper may now be specified. These Hs, their gener
definition, and rationale for selection

are listed below. In parentheses, following the definition, is the manifestation of the H as it appears in the raw data.

1. Position Preference: consistent response to one position. This H has been frequently observed in monkey and in rat behavior (*III*).

2. Position Alternation: alternating between positions from trial to trial. While this H has been noted only rarely in the monkey, Gellermann (1933) felt that it might occur in sufficient degree to control for it. It is a common phenomenon in rats (*IOI*).

3. Stimulus Preference: consistent response to one of the stimulus objects irrespective of reinforcement. This too has been commonly found in the primate and other orders (+ + + or − − −).

4. Stimulus Alternation: alternating between stimuli from trial to trial. This, like its position analogue, is uncommon in the monkey. It has, however, been shown to occur in other orders (+ − + or − + −).

5. Win-stay-Lose-shift (with respect to position): repetition of a response to a position which has just been rewarded and alternating away from a position not rewarded. This type of H has been noted in human behavior by Goodnow and Pettigrew (1955), and in monkey behavior by Harlow (1950) in a slightly different form under the name "differential cue" ($I + I$ or $I - O$).

6. Lose-stay-Win-shift (with respect to position): repetition of a response to a position not rewarded on the preceding trial, and alternation away from a position just rewarded. This is the reverse of the H above, and would not be expected to occur except in *S*s with special experimental histories. It was included in order to provide an H which one would expect to have zero strength ($I - I$ or $I + O$).

7. $(\text{Problem-solution behavior})_2$ or Win-stay-Lose-shift (with respect to the object): repetition of a response to an object which has just been rewarded, and alternation away from an object not rewarded. This H yields maximum reward in the learning-set situation, since correct responding begins on the second trial of the problem (+ + +, or − + +).

8. $(\text{Problem-solution behavior})_3$: manifestation of the correct response on the third trial although not on the second trial. This H is required by the well-known fact that the correct response may suddenly appear on later trials (+ − + or − − +).

9. Random Responding: this H, while it has a single form of manifestation, may be produced in any one of three ways: (*a*) The determinants of a response may fluctuate nonsystematically from trial to trial, in which case all sequences would be expected to occur with approximately the same frequency. (*b*) The *S* may be systematically responding to stimulus changes which are unrecorded and which are randomly related to the position sequence of the rewarded object. In this case, again, all response sequences would have equal expected frequencies. (*c*) Under certain special conditions Hs which have been ignored will contribute to this Random Responding H without affecting the estimation of any other H. These conditions are described elsewhere (Levine, 1959). For now it is sufficient to note that the Random Responding measure is conceptualized as an estimate of both nonsystematic responding and residual sequence strength. (This H is manifested in all sequences.)

These nine Hs, their definitions, and manifestations are summarized in Table 1. In the last column of this table are the symbols which will be used to represent the probability of occurrence of an H or the proportion of problems showing the H in a set of data. For example, the result $a = 0.5$ means that *S*s show Position Preference on 50% of the problems. The first goal of this paper is to demonstrate a technique for evaluating these probabilities.

Two further assumptions will be useful in attaining this goal. The first is that the Hs are mutually exclusive. The occurrence on a single problem of the sequence + + + may mean, among other possibilities, that either Stimulus Preference or $(\text{Problem-solution})_2$ has taken place, not both. In general, if a sequence may be attributed to two or more Hs and several instances of the sequence have occurred, different Hs may have been operating on different problems, but two Hs never combine on the same problem. The second assumption is that the above set of Hs includes all Hs whose probability is greater than

TABLE 1

DEFINITION OF THE HS EVALUATED IN THE PRESENT PAPER

H	Definition	Manifestation	Probability Symbol
Position Preference	Sequence of response to one side	*III*	a
Position Alternation	Alternating between positions on consecutive trials	*IOI*	b
Stimulus Preference	Sequence of responses to one of the relevant stimuli	$+++$ or $---$	c
Stimulus Alternation	Alternating between the relevant stimuli on consecutive trials	$+-+$ or $-+-$	d
Win-stay-Lose-shift (pos.)	Response to the position rewarded on the preceding trial	$I+I+I$ or $I+I-O$ or $I-O+O$ or $I-O-I$	u
Lose-stay-Win-shift (pos.)	Response to the position not rewarded on the preceding trial	$I+O+I$ or $I+O-O$ or $I-I+O$ or $I-I-I$	v
$(\text{Problem-solution})_2$ or Win-stay-Lose-shift (obj.)	Learning manifested on the second trial of a problem	$+++$ or $-++$	p_2
$(\text{Problem-solution})_3$	Learning first manifested on the third trial of a problem	$+-+$ or $--+$	p_3
Random Responding	Responses uncorrelated with recorded stimulus changes	All sequences	R

zero. These two assumptions permit the following statement:

$$a + b + c + d + u + v + p_2 + p_3 + R = 1.00 \quad [1]$$

THE EVALUATION OF THE H STRENGTHS

The assumptions outlined above determine the relationship between a set of data and the probabilities of the Hs. Figure 1 shows a set of 32 cells, each one of which represents one of the 32 possible sequences which can occur in a three-trial problem. The 32 cells are organized into two blocks of 16 each, the block on the left containing those sequences with a first trial minus, the other containing those sequences with a first trial plus. The rewarded position sequences are on the rows and the response sequences are on the columns.

Any one sequence may be a manifestation of some Hs and not of others. For example, $A-_1A-_2A-_3$ may be interpreted as Position Preference, Stimulus Preference, Lose-stay-Win-shift (with respect to position), or Random Responding, but not as any of the other five Hs. If, when E presents AAA, any of the other five Hs should be occurring, then $-_1-_2-_3$ cannot occur. In the $A-_1A-_2A-_3$ cell are listed the probability symbols associated with the four possible Hs. In general, each cell contains the symbols for the Hs which may produce the indicated sequence. The only new symbol is r. This is used here instead of R to represent Random Responding, and is defined by $r = R/4$. The justification for this change of variable is that the Random Responding H enters into four times as many cells as any of the other Hs. If $a = 1$, for example, a's influence is felt in only eight cells whereas if $R = 1$ its influence is distributed over 32 cells.

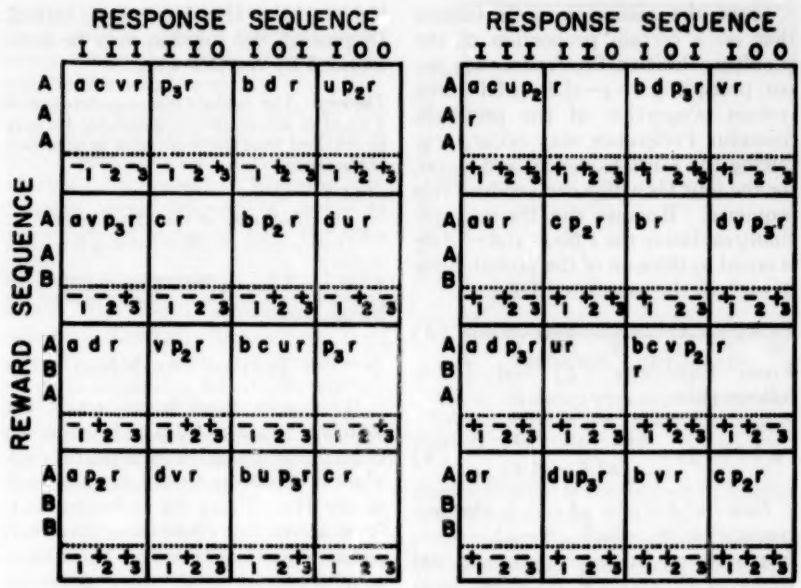

FIG. 1. Thirty-two cells representing the 32 possible sequences which can occur in a three-trial problem. The symbols in each cell represent the Hs which can produce the associated response sequence.

Its effect on any one cell would be $\frac{1}{4}$ the corresponding effect of any of the other Hs.

In a three-trial-per-problem learning-set experiment the data may be analyzed into the frequencies with which each of these 32 sequences occurs in a block of problems, and these frequencies may be used to estimate certain conditional probabilities. In particular, the probability of the outcomes on trials two and three given the reward sequence and the outcome on trial one may be estimated. For the $A-_1A-_2A-_3$ sequence, such a probability is symbolized as $P(-_2-_3|A-_1AA)$, where the vertical line is defined as the word "given." These probabilities may be obtained from the data by use of a few elementary probability theorems (Feller, 1950). To continue with the example selected,

$$P(-_2-_3|A-_1AA) = \frac{P(A-_1A-_2A-_3)}{P(A-_1AA)} = \frac{\frac{n(A-_1A-_2A-_3)}{N}}{\frac{n(A-_1AA)}{N}}$$

where $n(A-_1A-_2A-_3)$ and $n(A-_1AA)$ mean the number of times that $A-_1A-_2A-_3$ and $A-_1AA$, respectively, have occurred, and N is the total number of problems presented. The statement may be simplified to yield

$$P(-_2-_3|A-_1AA) = \frac{n(A-_1A-_2A-_3)}{n(A-_1AA)} \quad [2]$$

From the assumptions, it follows that on a certain proportion of the problems Position Preference may occur, producing $(-_2-_3|A-_1AA)$, on a certain proportion of the problems Stimulus Preference may occur, producing the same sequence, and so on, for the four Hs which can produce this sequence. Because the Hs are mutually exclusive the $P(-_2-_3|A-_1AA)$ is equal to the sum of the probabilities of each of the associated Hs, i.e.,

$$P(-_2-_3|A-_1AA)=a+c+v+r \quad [3]$$

From Equations [2] and [3] it follows that

$$a+c+v+r=\frac{n(A-_1A-_2A-_3)}{n(A-_1AA)} \quad [4]$$

Now, $n(A-_1A-_2A-_3)$ is the frequency with which $A-_1A-_2A-_3$ has occurred in the experiment and $n(A-_1AA)$ may be obtained from the left-hand matrix of Fig. 1 as the row total of the row containing $n(A-_1A-_2A-_3)$. That is,

$$\begin{aligned} n(A-_1AA)=&n(A-_1A-_2A-_3)\\ &+n(A-_1A-_2A+_3)\\ &+n(A-_1A+_2A-_3)\\ &+n(A-_1A+_2A+_3) \end{aligned}$$

The right side of [4] is, therefore, a number obtainable from the data, and Equation 4 is an equation in four unknowns.

With nine unknowns to be evaluated, nine such equations are required for simultaneous solution. These equations may be obtained from any nine cells yielding nonparallel equations. If the assumption is made that a given H has the same strength no matter in which cell it appears, then the solutions from the nine equations will give estimates of the nine H strengths for the block of problems considered. The first problem, then, that of evaluating the strengths of the various Hs, has been solved. In general, the solution may be summarized by the following:

Theorem: The probabilities associated with a set of m independently specifiable Hs may be obtained from the solution of m equations of the form

$$(j+k+\cdots)_i = \frac{\text{(frequency of the } i\text{th sequence)}}{\text{(frequency of the corresponding row total)}}$$

where $(j+k+\cdots)_i$ represents the sum of the probabilities of the Hs which may produce the ith sequence.

A TEST OF THE MODEL

It is now possible to apply the analysis to data from a learning-set experiment and to determine, for each block of problems, the relative strength of the Hs. Using the technique outlined above, one could solve three sets of nine equations based on 27 different sequences and obtain three estimates of each H. If the 27 sequences were carefully selected, the remaining five sequences could contribute another estimate of five of the Hs. Averaging the estimates for each H would yield a mean estimate of each of the H probabilities based on all the data. There is, however, one important objection to this procedure: application of the method without some prior testing of the model would leave the validity of the estimates in question. Some indication is first required that the estimates are a product of the behavior, and not simply of the mathematical machinery.

The present section deals with such validation. The test consists in estimating the probabilities from some of the sequences and then in predicting, on the basis of these estimates, the frequencies with which each of the remaining sequences should occur. These frequencies may be predicted from Equation 4 once the H probabilities are known. This frequency

prediction was applied to four sets of data, with a slightly different treatment for each. Each of these sets will be discussed in turn.

The first application was to the first block of three-trial-per-problem data from Levine, Levinson, and Harlow (in press). Eighteen sequences were selected in order to have two estimates of each H. The criterion for selection of each sequence was that it be based on no more than three Hs. Both sequences based only on Random Responding (i.e., $A+_1A+_2A-_3$ and $A+_1B+_2A-_3$), all eight sequences based only on two Hs, and eight of the sequences based on three Hs were selected. Averaging the solutions from the two sets of nine equations yielded:

$$a=0.08, \quad b=0.03, \quad c=0.16,$$
$$d=0.02, \quad u=-0.02, \quad v=-0.01,$$
$$p_2=0.20, \quad p_3=0.07, \quad R=0.44$$

A few interesting aspects of these results may be noted. First, the Lose-stay-Win-shift H was included with the prediction that $v = 0$, and a close estimate of zero was obtained. Second, b, d, u, and v all appear to be estimates of zero. This finding is not of special significance here, but will be referred to later because of its consistent emergence. Third, Equation 1 was not used in obtaining these estimates and may serve as a prediction. The sum of the obtained estimates is 0.97.

The values for these nine Hs were substituted into the equations for each of the remaining 14 sequences, and the predicted frequency for each sequence was obtained. The observed and predicted frequencies are plotted in Fig. 2 with each axis in the same units, so that perfect prediction would yield 14 points lying along the 45° line. It will be seen that the points distribute themselves in reasonable fashion along this line.

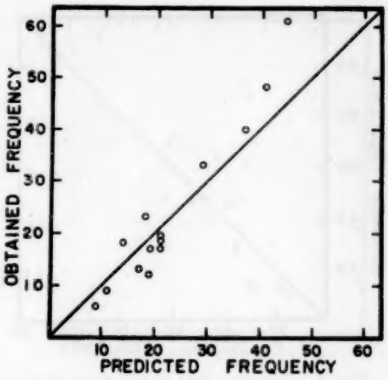

FIG. 2. Sequence frequencies from Block I (Problems 1–96) of the Levine et al. experiment.

In order to quantitatively describe the accuracy of prediction, a statistic describing the proportion of variance explained (PVE) by the predicted frequencies was devised. This is given by:

$$\text{PVE} = 1 - \frac{\sigma_{o.p}^2}{\sigma_o^2} \qquad [5]$$

where σ_o^2 is the variance of the observed values, and $\sigma_{o.p}^2$ is the variance around the 45° line, i.e., $\sigma_{o.p}^2 = \sum^n (o - p)^2/n$. The symbols o and p represent the observed and predicted frequencies, respectively, and n represents the number of points. For these data, PVE = 0.85.

The second set of data to which this test was applied was from the first 100 problems of a learning-set study by Harlow, Rueping, and Mason (in press). A new criterion was used in selecting the sequences for solution. The H probabilities were obtained primarily from sequences with a third trial minus, and predictions were made primarily for the sequences ending in

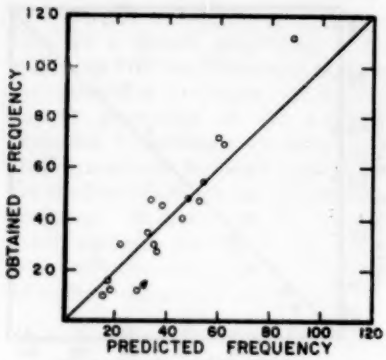

FIG. 3. Sequence frequencies from Block I (Problems 1–100) of the Harlow et al. experiment.

third trial plus. To accomplish this, it was convenient to use eight Hs, and so here it was assumed that $v = 0$. Twelve sequences ending in a third-trial minus, and two sequences with a third-trial plus (the two based only on p_3 and r) were selected to solve for all Hs but p_2. This H was then obtained from Equation 1. As a result, predictions could be made for 14 third-trial-plus sequences, and four third-trial-minus sequences. These 18 points are plotted in Fig. 3, where the PVE = 0.84. The specific probability values are:

$$a=0.13, \quad b=0.00, \quad c=0.19,$$
$$d=-0.03, \quad u=0.02, \quad p_2=0.13,$$
$$p_3=0.12, \quad R=0.44$$

Again b, d, and u appear to be estimates of zero. This finding, along with the assumption that $v = 0$, repeats the earlier finding.

The test was next applied to the first block of problems from a study by Schrier (in press). The same method of selection used in the preceding study was employed here in determining the sequences for solution, i.e., as many third-trial-minus sequences as necessary, two third-trial-plus sequences and Equation 1 were used. There was one minor difference, however. Schrier did not permit the reward to perseverate on one side for three consecutive trials. In this experiment, therefore, the top row of eight cells (see Fig. 1) does not exist, leaving only 24 sequences. In order to increase the number of sequences for which predictions could be made, it was assumed that $b = 0$, $v = 0$. This permits prediction for 12 sequences: 10 third-trial-plus and 2 third-trial-minus sequences. The results are shown in Fig. 4. Here the PVE = 0.91. The specific H probabilities are: $a = 0.13$, $c = 0.07$, $d = 0.00$, $u = -0.01$, $p_2 = 0.32$, $p_3 = 0.14$, $R = 0.36$. Again, d and u are estimates of zero, and the assumption that $b = 0$, $v = 0$ did not serve to reduce the PVE.

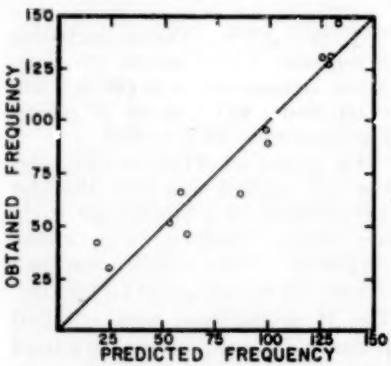

FIG. 4. Sequence frequencies from Block I (Problems 1–80) of Schrier's experiment.

The PVE, in fact, is somewhat larger in this study than in the preceding two sets of data. The difference is not so great as to warrant extensive speculation, but it was also noted that in the Schrier data R was lower than in the preceding sets. A check on whether variance and Random Responding were related entailed

applying the analysis to another block of data, the last 100 problems of the learning-set study described above by Harlow et al. This block of problems was selected because it became known in subsequent work that its R value was 0.20. Precisely the same analysis was applied as with the first 100 problems for this group. The results are shown in Fig. 5. The resulting PVE was 0.91 which bears out the possibility of the relationship. More relevant to the present topic, it provides another confirmation of the applicability of the model by this test, and suggests, along with the finding that R decreases throughout an experiment (to be described in the next section), that the PVEs would have been even higher had the test been applied to last blocks rather than first blocks of problems.

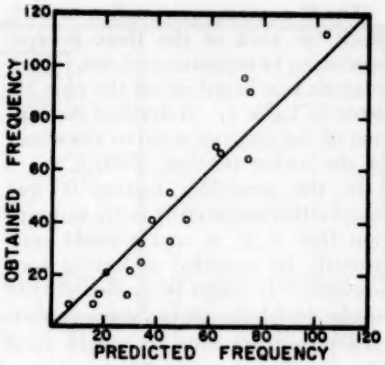

FIG. 5. Sequence frequencies from Block VI (Problems 501–600) of the Harlow et al. experiment.

AN ANALYSIS OF LEARNING-SET FUNCTIONS INTO H COMPONENTS

Now that the model has been described and its correspondence to behavior has been indicated, the analysis may be applied to demonstrate its value in describing behavioral processes. Harlow, Rueping, and Mason (in press) have performed a learning-set experiment with young rhesus monkeys in which they compared the effects of age upon the acquisition of learning set. Three groups of 10 *S*s each received 600 problems starting at 60, 90, or 120 days. The learning-set functions showing percentage correct on Trial 2 for blocks of 100 problems are plotted in Fig. 6. By t test, the 60- and 90-day groups are not significantly different but the 120-day group is significantly different from both of these.

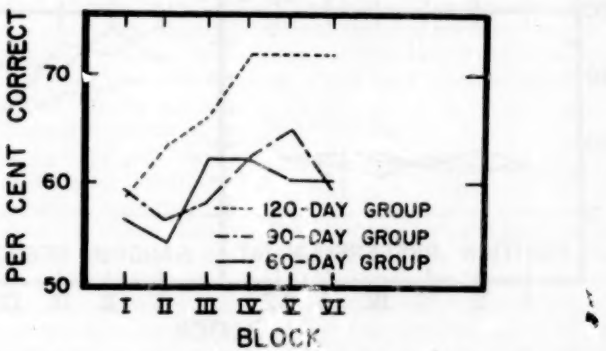

FIG. 6. Performance of the three groups in the Harlow et al. experiment as measured by the percentage of correct responses on Trial 2 for each 100-problem block.

The H analysis was applied to each block for each of the three groups, producing 18 separate analyses. Each analysis was based on all the nine Hs listed in Table 1. A detailed description of the analysis is given elsewhere by the author (Levine, 1959).

In the preceding section it was found either empirically or by assumption that b, d, u, and v could consistently be regarded as having zero strength. It might be predicted that in the 18 blocks of problems considered here these four Hs would yield 72 estimates of zero. These 72 estimates ranged from -0.05 to $+0.05$. The mean was at -0.002 with $\sigma = 0.02$. This finding further validates the model by confirming an expected finding, and simplifies the Harlow et al. data by eliminating four of the Hs from consideration.

The remaining Hs are plotted in Fig. 7. The p_2 and p_3 values have been added together to depict the number of problems manifesting a learning H, and are plotted in the upper left-hand quadrant. The differences here reflect the differences seen in Fig. 6, that the 120-day group shows more problem-solution behavior throughout than the 60- and 90-day groups.

Consideration of the remaining three Hs suggests some of the sources of these differences. The Position Preference H, plotted in the lower left-hand quadrant, is striking in that this H has the same strength for the three groups. Thus, the differ-

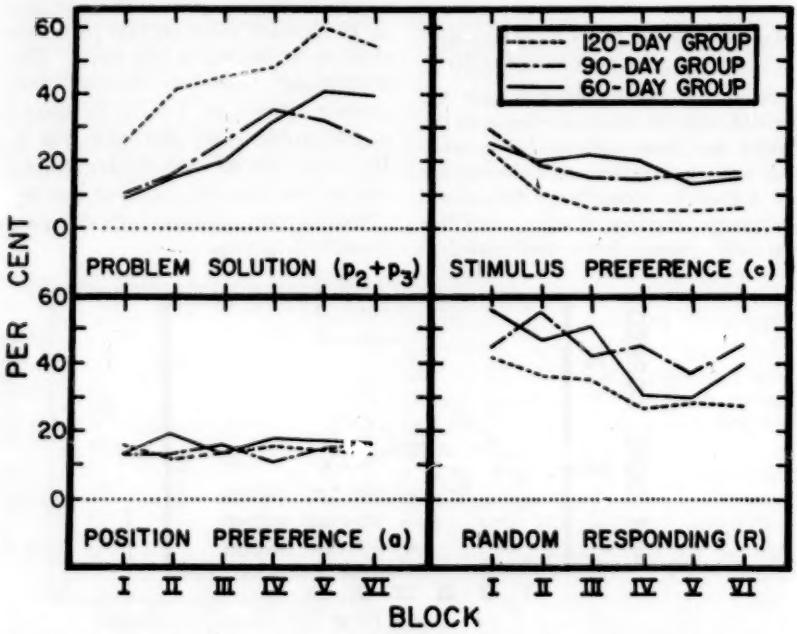

FIG. 7. Analysis of the three Harlow et al. groups. Each quadrant shows the percentage of problems in which the behavior was determined by the indicated H.

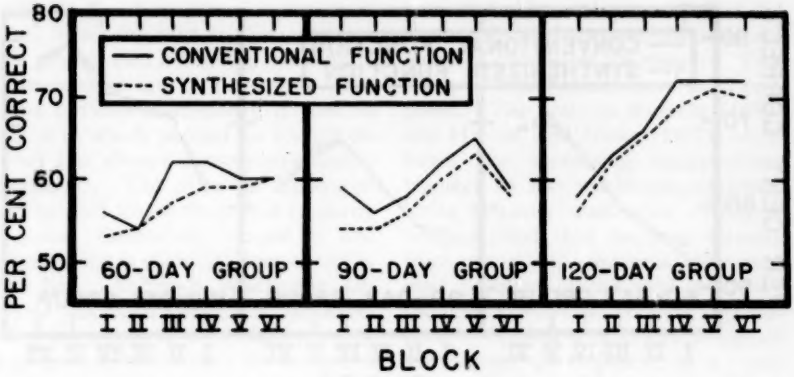

FIG. 8. Conventional learning-set functions and synthesized learning-set functions for the Trial-2 data of the three Harlow et al. groups.

ence in over-all performance between the 120-day and the 60- and 90-day groups is not attributable to this H. The basis for the difference between the learning-set functions may be seen in the right-hand side of Fig. 7. This difference is attributable specifically to the differences in Stimulus Preference and Random Responding.

In addition to providing more detail about the differences among the three groups, Fig. 7 shows the various behavioral processes which underlie the learning-set functions. Position Preference is again unique in that it does not decrease but has a constant strength at about 0.18. A comparison of this H with the other two nonsolution Hs shows that Hs do not extinguish simultaneously or at the same rate. Also, the $p_2 + p_3$ plot shows the progress of learning "purified" of the other behavior patterns. It may well be, although this is clearly conjecture at present, that the formulas describing these elementary processes will have greater simplicity and universality than the formulations of what is here seen as a complex, composite function: *the* learning curve.

Since it is clear that these processes underlie the learning-set function, it should be possible to synthesize this function by proper combination of the H values. Two classes of learning-set functions may be considered. One is the class described in Fig. 6, percentage correct on Trial 2; the other is percentage correct on Trial 3. Percentage correct on Trial 2 may be synthesized from the following considerations: whenever $(\text{Problem-solution})_2$ occurs, the S is rewarded on Trial 2; when $(\text{Problem-solution})_3$ occurs, the S is never rewarded on Trial 2; and whenever any of the other Hs occurs, the S is rewarded half of the time on Trial two. Percentage correct on Trial 2 would then be estimated by

$$\%\ \text{Correct} = \left[p_2 + \frac{1-(p_2+p_3)}{2}\right] \times 100 \quad [6]$$

The estimates for each block of problems, as well as the conventional learning-set functions, are plotted in Fig. 8, where it will be seen that there is a good fit with two kinds of deviations. The first is that the synthesized values consistently underestimate the conventional values by

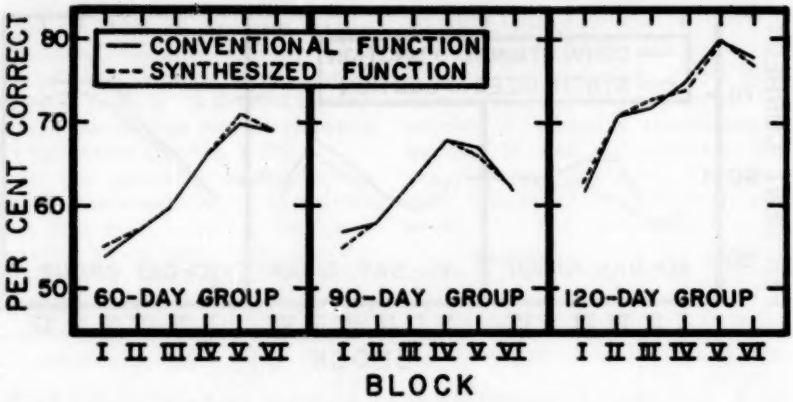

FIG. 9. Conventional learning-set functions and synthesized learning-set functions for the Trial-3 data of the three Harlow et al. groups.

about 2%; the other is that the synthesized curve tends to be a more regular learning function. For example, the strange decrements seen in the empirical functions for the 60- and 90-day groups are either eliminated or decreased. Whether either or both of these characteristics are reliable is, for the present, an empirical question. It has not been proved that they must appear.

The trial-three learning-set functions may be synthesized by considering that each time $(\text{Problem-solution})_2$ or $(\text{Problem-solution})_3$ occurs the S is rewarded on Trial three, and whenever any of the other Hs occurs the S is rewarded half the time. Percentage correct on Trial 3 would then be estimated by

$$\%\text{-Correct} = \left[p_2 + p_3 + \frac{1-(p_2+p_3)}{2}\right] \times 100 \quad [7]$$

The synthesized and conventional Trial-3 learning-set functions are plotted in Fig. 9, where it will be seen that the two functions are identical. This identity does not have the status of a verified prediction since it has been demonstrated (Levine, 1959) that the operations employed in obtaining the two sets of functions are virtually identical. However, the correspondence indicates that the assumptions of the model are reasonable in that they do lead to these operations.

DISCUSSION

The model presented here has a few useful features. Primarily, it provides a technique for evaluating the strengths of a variety of Hs. This method follows directly from certain relatively simple assumptions concerning the appearance of response patterns. Secondly, almost all of the variance of the frequencies of response sequences occurring in a block of problems may be explained by the model (Figs. 2–5). Then, evaluation of H strengths at successive stages of learning shows that Hs follow regular functions (Fig. 7). Finally, the conventional learning-set functions may be synthesized by appropriate combination of the Hs (Figs. 8–9).

While the data are, for the most part, in accord with predictions from the model, two intriguing systematic discrepancies are to be noted. The most obvious discrepancy is seen in Fig. 8 in which percentage correct on Trial 2 is always consistently underestimated. The other is illustrated in Figs. 2–5 where the points typically organize themselves around a line whose slope is slightly larger than the 45° line. Although insufficient analysis has been performed to determine the source of these discrepancies, a promising lead is that the sequences $-+-$ and $++-$ are occurring somewhat more frequently than would be predicted by the model. It may be shown that both discrepancies would follow if this were the case. These discrepancies may mean that some H has been overlooked or that some of the other assumptions require revision.

Assuming that the model in its current form is approximately correct, it provides interesting implications for some current psychological problems. The central property of the model is that it takes as the dependent variable unit the H, defined as a pattern of responses to selected stimuli. The specification of a behavior pattern means that this pattern as a whole is susceptible to the traditional effects of reinforcement operations, i.e., it is possible to reinforce some Hs and extinguish others. Thus, one may reinforce Position Alternation, Stimulus Alternation, or various kinds of stay-shift combinations. This idea of the reinforcement of a response pattern has specific relevance for understanding the phenomenon of learning-to-learn as seen in the discrimination learning-set situation. Since Harlow's (1949) demonstration that *S*s show progressive improvement on Trial-2 performance in successive problems the question of the nature of this improvement has provided a challenge to theory. Two types of explanations have been proposed. The first, by Riopelle (1953) and Harlow and Hicks (1957), attributes the increasing interproblem transfer to the weakening of interfering response tendencies. Riopelle demonstrated that negative transfer from preceding problems decreased over problem blocks, and described the phenomenon as "transfer suppression." Harlow and Hicks described learning-set development as a "unitprocess" which, they suggested, is an inhibition of incorrect behaviors. These interpretations omit the conception of reinforcement as directly strengthening any response.

The second explanation, by Restle (1958), accepts the strengthening of a response via reinforcement during the experiment, but requires no assumption about the character of the response. Restle, rather, postulates a class of stimuli, "Type-a cues," responses to which are always rewarded, and which become conditioned to the correct response during the course of the learning-set experiment.

The explanation of learning-set development suggested by the present paper is consonant with this aspect of Restle's theory. The explanation results from assuming that the *S* is capable of responding in terms of Hs, and that Hs may be reinforced. One of the Hs, Win-stay-Lose-shift (with respect to the object), is the H which the *E* chooses to reinforce. There is nothing unique about this H. It has the same formal status as responses to one position or any of the other Hs. Learning-set development then is the gradual strengthening, via 100% reinforcement, of this H, and the gradual extinction, because of 50% reinforcement, of the other Hs. Thus, this

example of learning-to-learn is amenable to treatment by any conventional reinforcement theory, so long as a *pattern* of response is taken as the dependent variable. Note that "Win" and "Lose" are analogous to Restle's Type-a cues, and that the responses are staying and shifting (with respect to the object), respectively.

The model provides description not only of the H which is reinforced but also of other Hs. An important empirical finding presented in this paper is that these Hs may not all extinguish at the same rate. This finding creates a problem for current learning theories, which make no provision for several extinction processes. It is, of course, too soon to know how many different extinction processes must be postulated and to develop theories about the nature of the differences. The analysis described herein should make a contribution toward solving these problems.

REFERENCES

FELLER, W. *An introduction to probability theory and its applications.* (2nd ed.) New York: Wiley, 1957.

GELLERMANN, L. W. Chance orders of alternating stimuli in visual discrimination experiments. *J. genet. Psychol.*, 1933, **42**, 207–208.

GOODNOW, JACQUELINE J., & PETTIGREW, T. F. Effect of prior patterns of experience upon strategies and learning sets. *J. exp. Psychol.*, 1955, **49**, 381–389.

GOODNOW, JACQUELINE J., & POSTMAN, L. Learning in a two-choice probability situation with a problem-solving setting. *J. exp. Psychol.*, 1955, **49**, 16–22.

GOODNOW, JACQUELINE J., SHANKS, BETTY, RUBINSTEIN, I., & LUBIN, A. What is the human subject responding to in a two-choice task? *WRAIR Problem-Solving Proj. Memo.*, 1957, No. 3.

HARLOW, H. F. The formation of learning sets. *Psychol. Rev.*, 1949, **56**, 51–65.

HARLOW, H. F. Analysis of discrimination learning by monkeys. *J. exp. Psychol.*, 1950, **40**, 26–39.

HARLOW, H. F., & HICKS, L. H. Discrimination learning theory: Uniprocess vs. duoprocess. *Psychol. Rev.*, 1957, **64**, 104–109.

HARLOW, H. F., RUEPING, R. R., & MASON, W. A. Discrimination, discrimination learning set, and delayed reaction. *J. comp. physiol. Psychol.*, in press.

KRECHEVSKY, I. "Hypotheses" versus "chance" in the pre-solution period in sensory discrimination learning. *Univer. Calif. publ. Psychol.*, 1932, **6**, 27–44.

LEVINE, M. A model of hypothesis behavior in discrimination learning set. Unpublished doctoral dissertation, Univer. of Wisconsin, 1959.

LEVINE, M., LEVINSON, BILLEY, & HARLOW, H. F. Trials per problem as a variable in the acquisition of discrimination learning set. *J. comp. physiol. Psychol.*, in press.

RESTLE, F. Toward a quantitative description of learning set data. *Psychol. Rev.*, 1958, **65**, 77–91.

RIOPELLE, A. J. Transfer suppression and learning sets. *J. comp. physiol. Psychol.*, 1953, **56**, 108–114.

SCHRIER, A. M. Comparison of two methods of investigating the effect of amount of incentive on performance by monkeys. *J. comp. physiol. Psychol.*, in press.

(Received March 27, 1959)

Psychological Review
Vol. 66, No. 6, 1959

ACTIVATION: A NEUROPSYCHOLOGICAL DIMENSION[1]

ROBERT B. MALMO

Allan Memorial Institute, McGill University

There have been three main lines of approach to the problem of activation: (*a*) through electroencephalography and neurophysiology, (*b*) through physiological studies of "behavioral energetics," and (*c*) through the learning theorists' search for a satisfactory measure of drive. Before attempting a formal definition of activation, I shall briefly describe these three different approaches to the concept.

Neurophysiological approach: Lindsley's Activation Theory.[2] The neurophysiological approach to activation had its origin in electroencephalography (EEG). Early workers in the EEG field soon discovered that there were distinctive wave patterns characterizing the main levels of psychological functioning in the progression from deep sleep to highly alerted states of activity (Jasper, 1941). In deep sleep large low-frequency waves predominate. In light sleep and drowsy states the frequencies are not as low as in deep sleep, but there are more low-frequency waves than in the wakeful states. In relaxed wakefulness there is a predominance of waves in the alpha (8–12 c.p.s.) range that gives way to beta frequencies (approximately 18–30 c.p.s.) when the *S* is moderately alert. Under highly alerting and exciting conditions beta waves predominate. In addition to the increased frequency of the waves under these conditions of heightened alertness there is also a change from a regular synchronized appearance of the tracing to an irregular desynchronized tracing, usually of reduced amplitude.

[1] Support for some of the research reported herein has come from the following sources: National Institute of Mental Health, National Institutes of Health, United States Public Health Service: Grant Number M-1475; Medical Research and Development Division, Office of the Surgeon General, Department of the United States Army: Contract Number DA-49-007-MD-626; Defence Research Board, Department of National Defence, Canada: Grant Number 9425-04; and National Research Council of Canada: Grant Number A. P. 29.

Grateful acknowledgment is made to A. Amsel, R. C. Davis, S. M. Feldman, P. Milner, M. M. Schnore, R. G. Stennett, D. J. Ehrlich and L. R. Pinneo for constructive criticism of the manuscript.

The main parts of this paper were presented in a Symposium entitled, "Experimental Foundations of Clinical Psychology," under the chairmanship of Arthur J. Bachrach, at the University of Virginia, April 1–2, 1959. To Ian P. Stevenson, who was the discussant of my paper on that Sympsoium, I owe a debt of gratitude for his very helpful comments.

[2] I am using neuropsychology in a rather broad sense, meaning to include the work often referred to by the term "psychophysiology." This usage implies that the chief problems being studied are psychological ones, and it also stresses the importance of neurophysiological techniques. It is true that, strictly speaking, many of the physiological techniques in use are not neurophysiological ones; yet our main interest lies in the central neural control of the physiological functions under study rather than in the peripheral events themselves.

Later on in the paper I shall attempt a formal definition of activation. For the first section of the paper, I believe that it will be sufficient to say that in using the term "activation" I am referring to the intensive dimension of behavior. "Arousal" is often used interchangeably with activation; and level of drive is a very similar concept. For instance, a drowsy *S* is low, an alert *S* is high in activation.

For Lindsley's theory, desynchronization (called "activation pattern") became the single most important EEG phenomenon. My use of the term "desynchronization" is purely descriptive. Desynchronization or "flattening" in the EEG tracing was consistently found associated with increased alertness in a large variety of experiments with animal and human *S*s. The consistency and generality of this phenomenon suggested the existence of mechanisms in the brain mediating behavioral functions having to do with levels of alertness, although at the time that the original observations were made it was not at all clear what these neural mechanisms were.

With the discovery of the ascending reticular activating system (ARAS), however, there was rapid and very significant advance in theory and experimentation. Some of the most important general findings have been as follows: (*a*) Lesions in the ARAS abolished "activation" of the EEG and produced a behavioral picture of lethargy and somnolence (Lindsley, 1957). (*b*) The "activation pattern" in the EEG was reproduced by electrical stimulation of the ARAS. Furthermore, in the monkey, Fuster (1958) recently found that concurrent ARAS stimulation of moderate intensity improved accuracy and speed of visual discrimination reaction. He also found that higher intensities had the opposite effect, producing diminution of correct responses and increase of reaction times. Interpretation of these latter findings is complicated by the fact that they were obtained with stimulation intensities higher than the threshold for the elicitation of observable motor effects such as generalized muscular jerks. It is not stated whether intensity of stimulation was systematically studied. In any event, these observations of deleterious effect from high intensity stimulation are of considerable interest because they are what might be expected according to the activation theory.

The activation theory as first stated by Lindsley (1951)—although introduced in the handbook chapter on emotion—was, from the outset, conceived by him to be broader than an explanatory concept for emotional behavior. The theory was elaborated by Hebb (1955) in an attempt to solve the problem of drives. With the continuous flow of new experimental data on the ARAS (Lindsley, 1957), this area of neuropsychological investigation appears to be heading toward an important breakthrough. I shall attempt to state very briefly the main points of the current theory, drawing upon the ideas of several authors. According to this theory, the continuum extending from deep sleep at the low activation end to "excited states" [3] at the high activation end is very largely a function of cortical bombardment by the ARAS, such that the greater the cortical bombardment the higher the activation. Further, the relation between activation and behavioral efficiency (cue function or level of performance) is described by an inverted **U** curve. That is, from low activation up to a point that is optimal for a given function, level of performance rises monotonically with increasing activation level, but beyond this optimal point the relation becomes nonmonotonic: further increase in activation beyond this point produces a fall in performance level, this fall being

[3] The expression "excited states" is frequently used to refer to the upper end of the activation continuum. In using this term I do not wish to imply increased overt activity. In fact, overt activity may be reduced to a very low level at the high end of the continuum, when—for example—a person is immobolized by terror.

directly related to the amount of the increase in level of activation.

Principles of neural action that could account for the reversal in the effects of nonspecific neural bombardment of the cortex by the ARAS have long been known (Lorente de Nó, 1939, p. 428). Circulation of neural impulses in a closed chain of neurons (or "cell assembly" to use Hebb's [1949] term) may be facilitated by impulses arriving outside the chain (e.g. from the ARAS). According to Lorente de Nó's schema, such extraneous impulses have the effect of stimulating certain neurons subliminally thus making it possible for an impulse from within the chain to finish the job, that is make it fire at the appropriate time in the sequence, when alone, without the prior hit, it would have failed to fire it.

Again, according to the same account by Lorente de Nó (1939, p. 428), the deleterious effects of overstimulation from impulses outside the chain can be explained. A neuron in the chain may fail to respond to stimulation if owing to repeated activity it acquires a high threshold, and this failure to transmit the circulating impulses would mean cessation of activity in a cell assembly. I proposed this kind of explanation previously (1958) to account for the downturn in the inverted **U** curve as an alternative to Hebb's suggestion that "the greater bombardment may interfere with the delicate adjustments involved in cue function, perhaps by facilitating irrelevant responses (a high *D* arouses conflicting ${}_{S}H_{R}$'s?)" (Hebb, 1955, p. 250).

It seems reasonable to suppose that as diffuse bombardment from the ARAS greatly exceeds an amount that is optimal for some simple psychological function being mediated by a particular cell assembly, the operation of that cell assembly will be impaired, and that the performance being mediated by it will suffer accordingly. This line of reasoning suggests that the inverted **U** relation should be found in quite simple psychological functions. Present evidence appears to support this suggestion. A recent (unpublished) experiment by Bélanger and Feldman, that I shall describe later in this paper, indicates that in rats the inverted **U** relation is found with simple bar pressing performance, and an experiment by Finch (1938) suggests that even such a simple response as the unconditioned salivary response yields the inverted **U** curve when plotted against activation level.

It may be noted that according to a response competition hypothesis, the inverted **U** relation should appear most prominently in complex functions where opportunities for habit interference are greater than they are in the case of simple functions. According to the response competition hypothesis, in the limiting case where response is so simple that habit interference is negligible, the relation between response strength and activation level should be monotonic. Therefore, finding the nonmonotonic relation in such simple responses as bar pressing and salivation raises strong doubts that the habit interference explanation can account for the seemingly pervasive phenomenon of the inverted **U** curve.

Principle of activation growing out of work on behavioral intensity. Even before the EEG work on desynchronization, the behavioral evidence had suggested the existence of some brain mechanism like the ARAS. The writings of Duffy (1951, 1957), Freeman (1948), and others of the "energetics" group have long stressed the importance of an intensity dimension in behavior.

In an attempt to obtain a measure

of this intensity variable, Duffy relied mainly on records of muscular tension (1932) while Freeman's favorite indicator was palmar conductance (1948). These workers concluded from their experiments that there was a lawful relationship between a state of the organism, called "arousal," "energy mobilization," "activation," or simply "intensity" and level of performance. Moreover they suggested that the relationship might be described by an inverted **U** curve(Duffy, 1957). This suggestion has proved heuristic as indicated by the current experimental attack on the inverted **U** hypothesis (Stennett, 1957a; Bindra, 1959; Cofer, 1959; Kendler, 1959).

The inverted **U** shaped curve has been shown to hold in numerous learning and performance situations where the amount of induced muscle tension was varied systematically (Courts, 1942). It is tempting to conclude that tension induction is simply one of the many ways to increase activation level, but as Courts' (1942) discussion suggests this conclusion would be premature. It is possible that squeezing on a dynamometer, a typical means of inducing tension in these experiments, may produce generalized activation effects as some data from Freeman indicate (1948, p. 71). But Freeman's data are insufficient to establish this point, and there are alternative explanations for the relationship between the performance data and induced tension (Courts, 1942). By repeating the induced-tension experiments with simultaneous recordings of EEG and other physiological functions it would be possible to determine how general the effects of inducing tension actually are. Such direct tests of the activation hypothesis are very much needed.

Drive and activation. A third approach to the activation principle was made by learning theorists, especially those of the Hull school. I have argued elsewhere (Malmo, 1958) that general drive (D), without the steering component, became identical in principle with activation or arousal. Set aside for the moment the attractive possibility of using ARAS as a neural model for mediation of D, and consider only the methodological advantages of physiological measures in the quantification of D. It seems that none of the other attempts to measure D have been really satisfactory, and that physiological indicants where applied have been surprisingly effective. Learning theorists up to the present time have made only very occasional use of physiological measures. For instance, in arguing that a previously painful stimulus had lost its drive properties, Brown (1955) cited the absence of physiological reaction when the stimulus was applied. More recently, Spence (1958) has reported some success with physiological measures in his studies of "emotionally-based" drive.

In keeping with traditional views concerning the place of physiological measures in psychology, on those few occasions that they were employed at all they were applied to aversive or emotionally based drive. According to the activation principle, however, it should be possible to use physiological measures to gauge appetitionally based as well as aversively based drive. This means, for instance, that in a water deprivation experiment there should be close correspondence between number of hours of deprivation and physiological level. That is, heart rate, for example, should be higher in an animal performing in a Skinner box after 36 hours of deprivation than after 24, higher still after 48 hours of deprivation and so on. In my Nebraska Symposium paper I stated that, as far

as I was aware, this kind of experiment had not been reported (Malmo, 1958, p. 236).

Bélanger and Feldman in Montreal have recently completed such an experiment, and, as can be seen by inspecting Fig. 1, the results were as predicted by the activation hypothesis. Heart rate in rats showed progressive change corresponding with increasing hours of water deprivation. Although there were only seven rats in the group, this change in heart rate was highly significant. Deprivations were carried out serially on the same group of animals, commencing at 12 hours and proceeding to 24, 48 hours and so on with sufficient hydration (four to seven days) between deprivation periods to prevent any cumulative effects from affecting the experiments. Heart rate was picked up by means of wire electrodes inserted in the skin of the animals and was amplified and registered graphically by means of a Sanborn electrocardiograph. Particular care was taken to record heart rate under nearly the same conditions of stimulation each time, that is, when the animal was pressing on the lever in the Skinner box or during drinking from the dispenser immediately after pressing. Under these conditions it was not possible to obtain sufficient heart-rate data at the 12-hour deprivation interval. Testing the animal under constant stimulating conditions is a very important methodological consideration. Some exploratory observations indicated that heart-rate measurements taken in a restraining compartment did not agree with those taken under the carefully controlled stimulus conditions provided by the Skinner box. I shall return to this finding later on because, aside from its methodological importance, I believe that it has considerable theoretical significance as well.

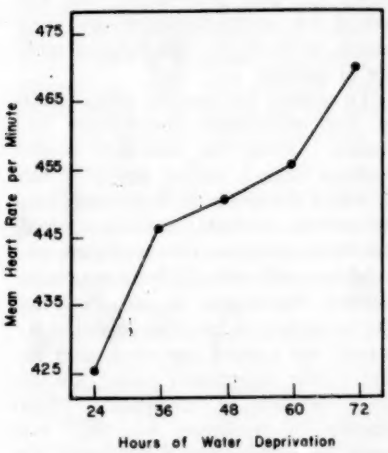

FIG. 1. Data from Bélanger and Feldman showing relation between water deprivation and heart rate in rats ($N = 7$). See text for explanation.

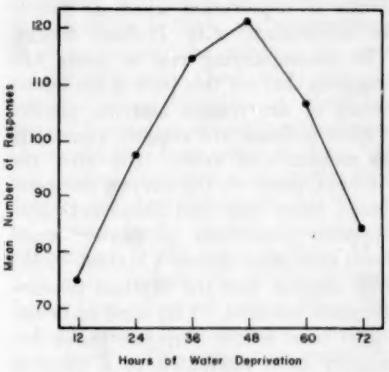

FIG. 2. Data from Bélanger and Feldman showing relation between water deprivation and Skinner box performance in rats ($N = 7$). See text for explanation.

Figure 2 presents the behavioral data which are again in remarkably good agreement with prediction from the activation hypothesis. Up to the 48-hour deprivation interval there is an increasing monotonic relationship between number of bar presses and

hours of deprivation which is strictly in accordance with Hullian theory. The accompanying rise in heart rate suggests that for this part of the curve, hours of deprivation and the physiological indicant are roughly equivalent as measures of drive. But after the 48-hour point on the curves, the combined heart rate and behavioral data support predictions previously made from activation theory (Malmo, 1958) and suggest that the Hullian position requires revision. This kind of downward turn in the response curve has usually been attributed to a physical weakening of the animal due to the deprivation of food or water. In the absence of physiological data such an assumption appeared reasonable in many cases, although it did not account for response decrement in certain experiments where physical weakening seemed to be ruled out (Finan, 1940; Freeman, 1940; Fuster, 1958; Kaplan, 1952; Stennett, 1957a). Attack on this problem with physiological methods should soon provide a definitive answer concerning the main determinants of this response decrement. The present experiment represents an important first step in a program of animal studies that should go a long way towards solving this problem. It is not claimed that this one experiment demolishes the inanition hypothesis, but it does seem that the results are opposed to it. Heart rate in the Minnesota starvation experiments was found lowered in the weakened individuals (Malmo, 1958, p. 252) whereas heart rate in the present experiment was markedly increased during the period when number of responses was declining. Moreover, Bélanger was careful to record the weights of the animals all through the experiments, and he observed only very slight changes in weight, even at the 72-hour deprivation interval. Again, it should be stressed that all through the experiment the animals received four to seven days of hydration between conditions. Furthermore, it is interesting to note that the animals continued to press the bar at fairly regular intervals in the high deprivation conditions (with response decrement). That is, their behavior did not appear as though they had "given up." The acts of pressing continued to occur regularly, only they were separated by longer temporal intervals than under more optimal conditions of deprivation.

The increasing monotonic curve for heart rate did not seem to be simply due to the physical conditions of exertion associated with the act of bar pressing. It is true that up to the peak of the performance curve increasing heart rate was accompanied by increasing frequency of bar pressing, but past this point, heart rate continued to show rise despite the decline in exertion due to bar pressing. One might conjecture that exercise may have had greater effect on heart rate under extreme deprivation, but this would be counterbalanced—to some extent, at least—by the reduced number of presses.

To control for possible serial effects in this experiment there were two checks. First, he obtained similar findings from a second group of rats in which the order of deprivation conditions was reversed, commencing with the 72-hour deprivation condition, and finishing with the 12-hour condition. Second, the group of rats that had the ascending order of deprivation intervals were tested one week after the end of the experiment under the 60-hour deprivation condition. Mean number of responses was 96.7 and mean heart rate was 458.9 beats per minute, thus providing good agreement with the results that were obtained in the main experiment.

Finally, it is possible to speculate along various lines about how the heart rate data could be accounted for without involving the concept of activation. Obviously, further experimentation is needed, but it is encouraging nonetheless that the first animal experimentation specifically designed to explore the relation between appetitional drive and activation turned out according to prediction.

CHARACTERISTICS OF ACTIVATION

The three approaches described in the previous section appear to lead to the same fundamental concept of activation. It will, of course, be difficult to state a precise definition of activation that will satisfy everyone. Neurophysiologically oriented workers will maintain a healthy scepticism concerning the so-called "peripheral" indicants of activation. The "energetics" group while welcoming the extended use of what is essentially their own methodology will in company with some learning theorists look askance at theoretical models that verge on neurologizing. Despite differences in point of view, however, it seems worthwhile to attempt to deal with certain major characteristics of activation on which we may expect a large measure of agreement.

Activation level a product of multiple factors. When a man is deprived of sleep for some 60 hours his activation level appears higher than it was before he had suffered sleep loss. Physiological indicants reveal an upward shift in activation level that is gradual and progressive throughout the vigil (Malmo, 1958). Having once demonstrated these physiological changes it is tempting to dispense with physiological recording in further work, assuming that 60 hours of deprivation will invariably produce a heightened state of activation. Such an assumption, however, cannot be made. An example will make clear why this assumption is untenable. A sleep-deprived *S* requires constant stimulation to prevent him from going to sleep. It is a general finding in such studies that despite the best intentions of the *S* to remain awake he will "catnap" if left alone. When he is working at a task trying to keep his efficiency from falling, the effect of major sleep loss is to produce a large increase in activation level. The important point to see here, however, is that the higher activation level is a combined product of the stimuli and their demands on him plus the condition of sleep loss. Without such stimulation, the *S* would surely fall asleep and we know from our studies of sleep that physiological levels drop very rapidly as one drifts into sleep. It is obvious, therefore, that in the absence of the task, physiological indicants at 60 hours' deprivation would show lower, not higher, activation in comparison with the rested condition.

That the "drive state" is in large part determined by environmental stimulating factors is indicated also by the observations of Bélanger and Feldman in their water deprivation experiments. Incidental observations suggested that, in addition to being more variable, heart rates recorded from the animal in a restraining compartment seemed to be consistently lower than those that were recorded when the animal was pressing the lever or drinking. In the restraining compartment the animal could view the lever through glass so that apparently mere sight of the lever was insufficient stimulation to produce the full effect upon heart rate that was produced by the acts of pressing on the lever and drinking. It thus ap-

peared that, with deprivation time approximately the same, activation level differed appreciably depending upon the conditions of external stimulation. These observations were merely incidental ones in this experiment, and they should be repeated; but they encourage the point of view that activation level is in large part a function of environmental stimulating conditions. The experiments of Campbell and Sheffield (1953) seem to point in the same direction. In the absence of sufficient environmental stimulation, food deprived rats are no more active than satiated ones, but with stimulation they are much more active than the satiated controls.

Returning to the example of the water deprived rat in the Skinner box, the two major factors determining the level of activation in that situation are (*a*) the internal conditions produced by deprivation and (*b*) the environmental stimulating conditions. To restate a point previously made, level of activation does not seem to be simply determined by the condition of deprivation alone. This would mean that depriving an animal of water per se could not produce some direct effect on motor mechanisms such as a simple discharge into the cardiac accelerating mechanism, leading to increased heart rate. Instead of some direct effect of this kind leading immediately over to some observable effector action, deprivation appears to have a sensitizing effect that is undetectable (or latent). According to this view, when appropriate stimulation does occur, the previously latent effect of deprivation will show itself in the heart rate: within limits, the longer the period of deprivation the higher the heart rate. Furthermore, according to activation theory, the same central mechanism that increases heart rate also acts to increase bombardment of the cerebral cortex. As previously stated, this central mechanism is presumed to be the ARAS.[4]

What could be the means of sensitizing cells in the ARAS by a condition such as deprivation of water or food? If some hormone like epinephrine were released by deprivation, it is conceivable that this hormone could act to sensitize the ARAS cells in degree proportional to the amount of time that the animal had been deprived. As a matter of fact, hormonal sensitization of neural mechanisms is a currently active area of research (Saffran, Schally, & Benfey, 1955; Dell, 1958).

There are some real difficulties in defending the position that the ARAS is a unitary intensity-mediating mechanism, because the ARAS does not appear to be a homogeneous anatomical system. Indeed, as Olszewski (1954) has shown, these central brain stem structures appear very complex and highly differentiated. This unreassuring fact must not be forgotten, but neither should it be accepted as precluding the unitary function. As Lashley points out in the discussion of Olszewski's paper, structural differences are not reliable indices of function when unsupported by other evidence.

As a matter of fact, there is some important functional evidence which encourages the unitary view despite the structural complexity of the ARAS. Dell (1958) has found that: "Epinephrine does not activate selectively mammillothalamocingular systems, . . . but instead activates the ascending reticular system *en masse*, thus leading to a generalized cortical arousal" (p. 370). Control experiments showed

[4] It is very likely that the descending reticular activating system is involved here too, but, at the present stage of knowledge in this field, it does not seem wise to introduce further complications into the neuropsychological model.

that the activation effect was due to a direct action of the epinephrine at the reticular level and not to an effect on the cerebral cortex. Similar results have been obtained by Rothballer (1956).

Another kind of difficulty for the quantitative view would be posed by showing that patterned discharge from the ARAS to the cortex (not merely total quantity of discharge) was the crucial factor in supporting some behavioral action. Don't the effector patterns of standing, walking, and righting pose just such a difficulty? The relation of midbrain mechanisms to posture seems to be clearly one in which patterns of discharge from the midbrain are important. But the decorticate mammal (guinea pig, rabbit, cat, dog) in which the cortex of both hemispheres has been removed shows approximately normal postural and progressional activities (Dusser de Barenne, 1934, p. 229). Since the activation concept under review deals with bombardment of the cerebral cortex, it appears that these noncortically mediated response patterns fall outside of phenomena under present consideration.

I should add, finally, that my admittedly speculative suggestion concerning hormonal sensitization is by no means essential to the main point which is that the behavioral evidence clearly shows the effects of deprivation to be latent (i.e. unobservable) under certain conditions. Moreover, this stress placed on the latent effects of deprivation is not mere hairsplitting. In addition to being required for an explanation of the Montreal experiments, this concept of latent deprivation effects appears to account in large measure for the findings of Campbell and Sheffield (1953), and more generally for the failure of random activity to adequately serve as a measure of drive or activation (Malmo, 1958).

Activation and the S–R framework. As the product of interaction between internal (perhaps hormonal) conditions and external stimulating ones, activation cannot be very reasonably classified as either stimulus or response. This means that the physiological measurements that are used to gauge level of activation do not fit very well into the S–R formula. It is perhaps useful to think of these physiological conditions as part of O in the S-O-R formula (Woodworth & Schlosberg, 1954, p. 2).

The momentary physiological reaction to a discrete stimulus like the sudden rise in palmar conductance accompanying pin-prick is not of primary concern to us in our study of activation. This kind of S–R reaction, important as it undoubtedly is for investigating other problems, is of little relevance for the study of activation, compared with the longer lasting changes. As Schlosberg has put it to me in personal communication, in employing skin conductance to gauge level of activation, one observes the "tides" and not the "ripples." I do not mean to disparage studies that use physiological reactions as R terms in the strict S–R sense. It is just that in this paper I am concerned with physiological functions only insofar as they are related to activation.

It may be queried whether we are dealing with a needless and hairsplitting distinction by saying that activation is not a response. However, the kind of difference I have in mind appears quite distinct and useful to keep in mind, though it should not be stressed unduly. Basically, it is the same distinction which Woodworth and Schlosberg (1956) make when they draw particular attention to the dif-

ference between slow and rapid changes in skin conductance. As examples of rapid changes in skin conductance, there are the "GSRs" as R terms in conditioned responses, and in free association tests. Examples of slow skin-conductance changes, on the other hand, are the gradual downward drifts that occur over hours during sleep (see Fig. 4), the slow downward changes in skin conductance in *S*s as they become gradually habituated to an experimental situation (Davis, 1934; Duffy & Lacey, 1946), and (going up the activation scale) the progressive upward changes in conductance during a vigil (Malmo, 1958).

I would not deny that there are stimuli and responses going on in the physiological systems, but at the present time I see no way of identifying and handling them. It should be added, however, that this does not give one license to completely disregard the antecedents of physiological changes. For instance, if the hand of a sleeping *S* becomes hot by being covered with heavy bedclothing the local thermal sweating induced thereby will bring about a sudden rise in palmar conductance which has nothing to do with activation. Or sleep may by induced by certain drugs which have a specific stimulating effect on respiration, such that respiration rate will not fall during sleep as it usually does (see Fig. 5 for curve obtained under nondrug conditions). Furthermore, artifacts due to movement and postural shifts may prevent muscle potentials from serving as reliable indicants of activation level.

Limitations of the activation concept. I am not attempting to solve the problem of selection, i.e., the problem of finding the neurophysiological mechanisms that determine which cues in the animal's environment are prepotent in the sense of winning out over other cues in triggering off a pattern of effector action. This point seems clear enough, especially when it is stressed that activation has no steering function; and yet there is still the risk that some critics may misunderstand and state as one shortcoming of this theory that it does not adequately handle the problem of selection. The theory may be open to criticism on the grounds that it is limited, but it should not be criticized for failing to do something which it was not intended to do.

It will be noted that in general an attempt is made to raise theoretical questions that stand a good chance of being answered by available experimental techniques. Schematically, the experimental paradigm is as follows:

Activation level	:	Low	Moderate High
Expected performance level	:	Low	Optimal Low

It is important to stress that the measure denoted by "moderate activation level" has meaning only in relative (not in absolute) terms. That is, the level is "moderate" because it is higher than that of the low activation condition, and lower than the level of the high activation condition. Comparisons are invariably of the within-individual, within-task kind, which means that the level of activation which is found to be optimal for one task is not directly compared with the level of activation which is found to be optimal for a different task. Thus, at the present stage of theorizing, no attempt is made to deal with the question of whether tasks which differ in complexity, for example, also differ with respect to the precise level of activation which is optimal for each one. However, I have dealt elsewhere (Malmo, 1958) with the related question of response competition, suggesting an alternative to the response competition explana-

tion for decrement in performance with increased activation (or *D*).

Again, the theoretical formulations may be criticized for being too narrow. But it must be kept in mind that their narrowness is due to the close nexus between theory and experiment in this program. These formulations may also be criticized for an unjustifiable assumption in the postulation of a communal drive mechanism. One may well ask where the evidence is that proves the existence of a state of general drive. In dealing with this kind of question, it is essential to refer back to the outline of the experimental paradigm. The experimental induction of the three discriminable activation levels referred to in the outline depends upon the controlled variation of certain conditions in the *S*'s environment. The fact that by varying conditions as dissimilar as appetitional deprivations and verbal incentives it is possible to produce similar shifts in physiological indicants provides a sound basis for introducing the operationally defined concept of activation level that cuts across traditional demarcation lines of specific drives. All this, of course, does not constitute final proof for a communal drive mechanism. Certainly further data are required before it is even safe to conclude equivalence of drive conditions in the alteration of physiological levels, to say nothing of proving the existence of a communal drive mechanism.

INTERRELATIONS BETWEEN PHYSIOLOGICAL INDICANTS OF ACTIVATION

Criticism directed against physiological measures as indicants of activation usually involves one or both of the following points. The first objection is that intercorrelations between physiological measures are so low that it is unreasonable to consider their use for gauging a single dimension of behavior. A second objection is that activation properly refers to events in the brain and that the correspondence between these central events and what may be observed in such peripheral functions as heart rate, respiration, muscle tension and the like is not close enough to permit valid inferences from the peripheral events to the central ones. In the following section, I shall attempt to answer these criticisms.

Intra- and interindividual correlations among physiological indicants of activation. In an unpublished paper, Schnore and I have discussed certain misconceptions that have confused some critics of physiological methods. The most serious misunderstanding concerns correlations among physiological measures. It is true that *inter*individual correlations are low, but this fact is actually irrelevant insofar as using these measures to gauge activation is concerned. The important question is whether significant *intra*individual correlations are found in a sufficiently high proportion of individuals, and the answer appears to be yes (Schnore, 1959).

What the low *inter*individual correlations mean, of course, is that an individual in any given situation may have a heart rate that is high relative to the mean heart rate for the group, and at the same time have a respiration rate or a blood pressure that is low relative to the group mean. These findings are in line with the principle of physiological specificity that is now supported by several lines of evidence.[5]

[5] The general principle of physiological specificity states that under significantly different conditions of stimulation individuals exhibit idiosyncratic but highly stereotyped patterns of autonomic and somatic activation. I use the term *physiological specificity* as a generic reference to autonomic-response stereotypy (Lacey & Lacey, 1958) to symptom specificity (Malmo & Shagass,

Physiological specificity is a separate problem that is in no way crucial for the activation hypothesis. An illustration will make this clear. Take a rather extreme example of an individual with very *high* heart rate (say 95 when the mean for his group under specified conditions is 75) and very *low* palmar conductance (50 micromhos when the group mean is 100). In an experiment with varied incentive, in going from a low incentive to a high incentive condition this *S* will likely show an increase in heart rate from 95 to say 110 and an increase in palmar conductance from 50 to say 60 micromhos. The main point is that even though the *S*'s heart rate is already high compared with the mean for his group, it goes still higher (concordantly with palmar conductance) when the stimulating situation increases the level of activation. This is the kind of intraindividual correlation between physiological measures [6] that is required for gauging the dimension of activation and, to repeat, the evidence strongly indicates that the intraindividual correlations are sufficiently high for this purpose.

Relations Between Central and Peripheral Indicants of Activation

As previously noted, the pioneer EEG workers observed definite changes in EEG pattern accompanying major shifts in the conscious state of the *S*. Moreover, they recognized a continuum of increasing activation usually referred to as the sleep-waking-excitement continuum, just as other workers like Freeman (1948) and Duffy (1957) employing peripheral measures of palmar sweating and muscular tension recognized it. Among the early workers in this field, Darrow (1947) studied EEG and other measures simultaneously, but only very recently have techniques been made available that can provide the kind of quantitative EEG measurements required for critical comparisons along the activation continuum. That is, from simple inspection of the raw EEG tracing it is possible to see gross differences between sleeping and waking, or between a drowsy, relaxed state and one of extreme alertness. But for experiments on activation it is necessary to have an instrument that will reveal measureable differences for "points" lying closer to each other on the activation continuum. For example, it is essential to have a measure that will discriminate reliably between a moderately alert and a highly alert state. For such discriminations the method of inspection will not do, and a device for objective quantification of the wave forms is required.

Because of its complexity the EEG tracing has been difficult to quantify, and although gross differences in activation level could be detected by simple inspection of the tracing, this method was too crude for more detailed work. However, with the advent of EEG frequency analysers, quantification of the EEG looked promising because these analysers were designed to provide quantified EEG data for each of many different narrow frequency bands. Unfortunately, these instruments have not proved useful because of insufficient stability. In our laboratory we have been trying band-pass filters to provide stable quantification of various selected

1949), and to stereotypy of somatic and autonomic activation patterns (Schnore, 1959).

[6] It is not claimed, however, that all physiological measures are equally useful for the purpose of gauging activation level On the contrary, as Schnore's experiments have suggested, some measures appear superior to others, and eventually we may be able to select the most discriminating ones and thus improve our measurement (Schnore, 1959).

frequency bands in which we are primarily interested (Ross & Davis, 1958). Results thus far appear highly encouraging.

Data indicating relationships between EEG and other physiological functions. In a recent sleep deprivation experiment, we found that palmar conductance and respiration showed progressive rise during the vigil, indicating increasing activation with deprivation of sleep. In the same experiment we recorded EEG and, by means of a band-pass filter, obtained a quantified write-out of frequencies from 8–12 per second, in the alpha range. It will be recalled that the classical picture of activation is reduction in the amount of alpha activity. Therefore, what we might expect to find in this experiment is progressive decrease in the amount of alpha activity. As a matter of fact, this is exactly what was found (Malmo, 1958, p. 237).

As Stennett (1957b) has shown, however, the relationship between EEG alpha activity and other physiological variables is sometimes curvilinear. In the sleep deprivation experiments physiological measurements were taken under highly activating conditions and at this high end of the continuum further increase in activation seems invariably to decrease the amount of alpha activity. But at the lower end of the continuum with the *S* in a drowsy state, increased activation has the opposite effect on alpha activity. An alerting stimulus, instead of producing a flattening of the EEG tracing, will actually produce an augmentation of the alpha activity. This has sometimes been referred to as a "paradoxical" reaction, although it seems paradoxical only when it is assumed that the relation between activation level and alpha amplitude is a decreasing monotonic one throughout the entire activation continuum. But Stennett (1957b) has shown that the relationship is not monotonic. From his data he plotted a curve which has the shape of an inverted **U**. From this curve it would be predicted that with a drowsy *S*, stimulation should *increase* alpha amplitude. From the same inverted **U** curve it would also be predicted that an *S* whose activation level was sufficiently high (past the peak of the curve) before stimulation would show a *decrease* in alpha amplitude. Actually, some unpublished experiments on startle by Bartoshuk fit these predictions very well.

Recent data indicate the usefulness of a 2–4 c.p.s. band-pass filter in experiments on sleep. The data in the figures that follow represent mean values from three men who slept all night in our laboratory after serving as *S*s in our sleep deprivation experiments.

Bipolar sponge electrodes, soaked in electrode jelly and attached to the *S* by Lastonet bands, were used for the parietal EEG placement (two thirds of the distance from nasion to inion, and 3 cm. from the midline on each side). The primary tracing was recorded by an Edin Electroencephalograph, and the two secondary tracings were integrations of the EEG potentials that were passed through band-pass filters for selective amplification of signals in the 2–4 and 8–12 c.p.s. frequency bands. Measurements on the secondary tracings were carried out with special rulers, and these measurements were converted to microvolt values by reference to calibration standards.

Method of recording and measuring palmar conductance was similar to that described by Stennett (1957a).

Electrocardiograms were picked up from electrodes placed on contralateral limbs, and heart rates were determined from measurements of electrocardiota-

chometric tracings. Respiration rates were obtained by means of a Phipps and Bird pneumograph.

All three *S*s slept well throughout the night (approximately from 10 P.M. to 9 A.M. after some 60 hours without sleep). Physiological recordings were carried out continuously during the whole period of sleep in each case, and except for occasional attention to electrodes (e.g. application of electrode jelly and saline to electrodes) the *S*s were undisturbed.

Four pairs of cellulose sponge electrodes were attached to the four limbs (to the pronator teres muscles of the arms and the peroneal muscles of the legs) for the purpose of recording muscle potentials. Primary muscle-potential tracings were recorded on the chart of a custom built Edin electromyograph (EMG). Electronic integrators (employing the condensor charge–discharge principle, like those used for the secondary EEG tracings), attached in parallel across the galvanometers of this EMG unit, integrated the muscle potentials over successive 4-second periods.

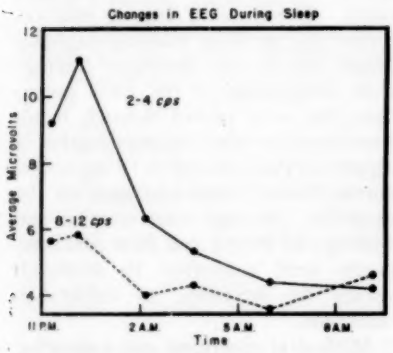

FIG. 3. Mean EEG values from three healthy young male *S*s during a night's sleep. Subjects had been sleep-deprived. Band-pass filters were used in connection with electronic integrators to provide quantitative data in the two different frequency bands.

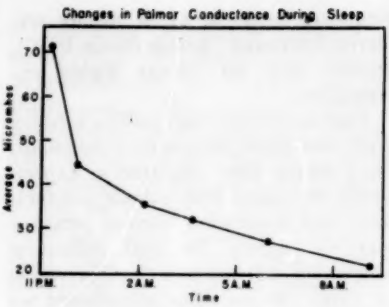

FIG. 4. Mean palmar conductance values from the same *S*s, at the same times during sleep as in Fig. 3.

These muscle-potential tracings were used to record movements and periods of restlessness during sleep. Five-minute periods free from muscle-potential activity and preceded by at least 5 minutes of movement-free tracings were chosen for measurement in order to provide the values plotted in Fig. 3–5. The actual times plotted on the baseline represent the medians for the three *S*s. In each instance the three times were close to one another.

In Fig. 3 observe that following a brief rise early in sleep the upper curve for 2–4 c.p.s. falls continuously during the entire period of sleep. This curve is consistent with published accounts of changes in EEG during sleep noted by inspection of the raw tracings (Lindsley, 1957, p. 68). Early in sleep there is an increase in slow waves around 2–4 cycles per second, but as sleep continues these waves are replaced by even slower ones. As far as I am aware, the data in Fig. 3 represent the first use of a 2–4 band-pass filter to quantify the EEG. The curve for 8–12 c.p.s. EEG also shows some fall, and the voltage is low in accordance with the well-known dis-

appearance of alpha waves from the raw tracings during sleep.

Figures 4 and 5 show data for palmar conductance, heart rate, and respiration, that were recorded at the same time as the EEG data. From the second plotted point on, there is rather close resemblance between these curves and the one for 2–4 c.p.s. EEG. It seems likely that a band-pass filter for fast frequencies in the beta range might yield a continuously falling curve commencing with drowsiness and continuing through the onset and early stages of sleep. There are serious technical difficulties in quantifying the next step of frequencies above the alpha band, but we are hopeful that a band-pass filter that has recently been constructed in our laboratory will overcome these difficulties.

Direct alteration of ARAS activity by means of electrical stimulation and related animal experimentation. The most relevant experiment on direct stimulation of the ARAS is, as far as I know, the one by Fuster (1958) that was mentioned earlier. By stimulating in the same part of the ARAS that produces the EEG picture of activation, Fuster was able to produce improved discrimination performance in the monkey. Presumably, this effect was achieved by causing a larger number of impulses from the ARAS to bombard the cortex. The assumption would be that before the onset of electrical stimulation the cortex was not receiving sufficient bombardment for optimal performance (Hebb, 1955) and that ARAS stimulation brought total bombardment in the cortex closer to the optimal value. The situation may not be as simple as this, but the success of the Fuster experiment encourages further experimentation along these same lines. Finding that level of performance can be altered by electrical stimulation of the ARAS opens up the exciting possibility that if amount of neural activity in the ARAS can be measured, we might find a direct correlation between a central measure of activation and level of performance. For instance, the Bélanger and Feldman experiment described earlier might be repeated with the addition of recordings from the ARAS. The aim of such an experiment would be to determine whether the continuous rise in the heart rate curve with increasing deprivation times could be matched by a similar rise in amplitude of deflections from recording in the ARAS with implanted electrodes. Recent neurophysiological experiments appear encouraging with respect to the feasibility of such an approach (Li & Jasper, 1953, pp. 124–125; Magoun, 1958, p. 68).

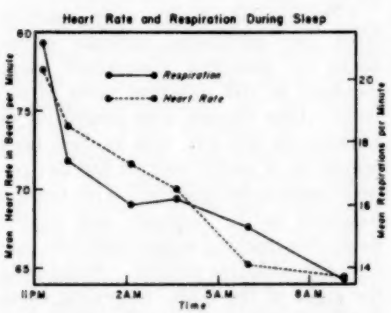

FIG. 5. Mean values for heart rate and respiration from the same *S*s at the same times during sleep as in Fig. 3 and 4.

EFFECTS OF INCREASED ACTIVATION ON LOCALIZED SKELETAL-MUSCLE TENSION IN PSYCHIATRIC PATIENTS

The implication of activation theory for various clinical phenomena might very well be the topic of a separate paper. Certainly there is not space to deal at length with the topic here. I have chosen, therefore, to present a

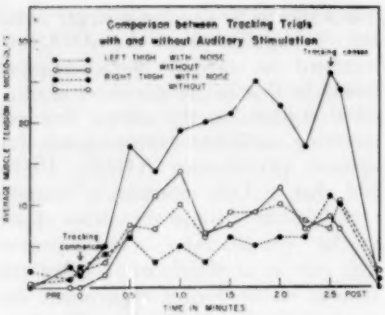

FIG. 6. Mean muscle tension from left thigh and right thigh from patient with complaint of tensional discomfort in the left thigh. Note that when patient was performing the tracking task under distraction (loud noise), tension rose in the left thigh but not in the right. See text for explanation.

few recent observations, chiefly in order to suggest how level of activation may be studied in relation to a clinical phenomenon.

The graph in Fig. 6 illustrates what appears to be a general finding in patients complaining of tensional discomfort in a localized muscular site. The data for the curves plotted in the figure were obtained from a psychiatric patient, a 42-year old woman who complained of muscular discomfort localized in the left thigh. In the session when these data were taken electromyograms (EMGs) were recorded from various muscles over the body; those from the left and right thighs are shown in the figure. The patient was engaged in pursuit tracking using an apparatus similar to the one employed by Surwillo (1955, 1956). Figure 6 shows that when a loud distracting noise, of the kind described by Schnore (1959), was presented during tracking, the tension in the left thigh was very much higher than that of the right thigh. When tracking was carried out under distraction free conditions this tensional difference between thighs was not observed.

Interpretation of these data seems quite straightforward. When level of activation was increased by presenting a loud distracting noise the effect was shown entirely in one muscle group, the left thigh, which was the symptom area in this patient. Simultaneous recordings of tension from other parts of the body showed that the tension was specific to the left thigh and was not merely increased on the whole left side of the body.

The specificity of the left thigh in indicating the higher activation is quite clear. Observe that tension in the thigh muscles on the opposite side of the body actually fell slightly under the activating condition.

The same procedure was carried out with a second patient, a young girl of 28, who complained of a distressing feeling of tightness in the neck on the right side. Results were similar to the ones obtained in the previous case, with activation again showing its effect specifically in the symptom area. When the loud distracting noise was turned on during tracking, tension in this area showed marked increase whereas tension in the muscles on the left side of the neck showed no rise whatever.

Very similar results were obtained from two additional patients whose areas of tensional discomfort were localized in still different parts of the body. One woman with complaint of tension on the left side of her neck served as a useful control for the patient previously described with tension localized in the opposite side of the neck. No tracking experiment was carried out with this patient. Apparently the sight of the EMG recording room for the first time was itself sufficient to increase the amplitude of muscle potentials from the symptom area so that

they become appreciably higher than those on the opposite side of her neck. The other woman (fourth patient in this series) complained of tensional discomfort that appeared to originate in the left shoulder. EMGs were recorded from the left and right shoulders of this patient while she lay in bed listening to the playback of a recorded interview. During the first part of the playback, tension was about the same on the two sides of the body. But when the topic concerning her dead sister commenced to come over the speaker, tension in the left shoulder became much greater than that in the right.

As far as could be determined, the EMG data from all these patients were consistent in suggesting that for skeletal-muscle tension in patients with well-developed tensional symptoms, increasing the activation level up to a certain point has the effect of raising muscle tension in one localized muscle group, the one in which the patient complained of tensional discomfort. It was not necessary for the patient to actually feel the discomfort during the experimental session for this differential result to appear. I have been using the term "symptom area" to refer to the muscle group where the discomfort was localized when present.

Interesting findings that appear to parallel those from the patients were obtained from three young male nonpatient *S*s in our recent investigation of sleep deprivation. As previously mentioned, evidence from EEG, palmar conductance, and respiration indicated that activation during tracking increased progressively with hours of sleep deprivation. In addition to these other physiological tracings, EMGs from various areas over the body were also recorded. One muscle area, a different one for each *S*, showed significant rise in tension over the vigil. It was the neck muscles in one *S*, the forehead in another, and the biceps muscle of the right arm in the third. In each case the one muscle showed statistically significant rise in tension, and in none of the *S*s was there significant tensional rise in any other muscle. In fact, there was regularly progressive and very significant fall in the tension of the left forearm in all three *S*s. As far as I know, none of the men actually complained of tensional discomfort in the areas showing rise in tension during the vigil.

Where high level activation is long continued as in a vigil or in certain psychoneurotic patients, it appears that skeletal tension may become localized to a single muscle group. The discomfort associated with this tension in some patients can become extremely severe. It should be noted that in one-session experiments, where rise in activation was for relatively short intervals of time, tensional rise occurred in more than one muscle group (Surwillo, 1956; Stennett, 1957a).

Methodologically, these results are important because they reveal a difference between EMGs and some other physiological measures with respect to gauging activation. Unlike heart rate or respiration rate that invariably yields one measure no matter how it is recorded, there are as many measures of muscle tension as there are muscles that can be recorded from. It appears that when sufficient care is taken, EMGs may be very valuable in helping to gauge activation, but that considerable caution is required in the interpretation of results, and especially in the interpretation of negative results.

From the clinical point of view it seems an interesting speculation that the patient's localized muscle tension may itself actually increase the general activation level. (I do not mean

the level of muscle tension all over the body.) Two main assumptions are involved in this suggestion. The first one is that the area of localized muscle tension in the patient acts like tension that is induced, for example, by having an *S* squeeze on a dynamometer. From the generalized effects of tension induction on learning and performance it is clear that the effects of increased muscle tension are quite general ones. Though crucial physiological data are missing in these experiments, as previously mentioned, one very likely explanation of these results is that the local increase in muscle tension somehow produces an increase in the general level of activation, with rise in heart rate and blood pressure, with fall in level of EEG alpha, and so on. This is the second assumption. The results of two recent experiments are in line with this assumption. Meyer and Noble (1958) found that induced tension interacted with "anxiety" in verbal-maze learning ("anxiety" measured by means of the MAS [Taylor, 1953]), while Kuethe and Eriksen (1957) in a study of stereotypy likewise reported a significant interaction between these two variables when "anxiety" was experimentally produced by means of electric shocks. The MAS appears to select individuals who are significantly above the mean in activation, and from the results of Schnore (1959) and Feldman (1958) it seems safe to conclude that anticipation of shock also leads to increased levels of physiological activity. In short, generalizing from the induced tension experiments, it seems reasonable to suppose that a patient's muscular tension in a small focal area might have the general effect of increasing activation. If such is the case symptomatic treatment might have significant general as well as specific effects. Although based on only one patient, Yates' (1958) results from symptomatic treatment of tics seems encouraging with respect to the feasibility of research in this general area.

SUMMARY

The neuropsychological dimension of activation may be briefly described as follows. The continuum extending from deep sleep at the low activation end to "excited" states at the high activation end is a function of the amount of cortical bombardment by the ARAS, such that the greater the cortical bombardment the higher the activation. The shape of the curve relating level of performance to level of activation is that of an inverted **U**: from low activation up to a point that is optimal for a given performance or function, level of performance rises monotonically with increasing activation level; but past this optimal point the relation becomes nonmonotonic: further increase in activation beyond this point produces fall in performance level, this fall being directly related to the amount of the increase in level of activation.

Long before the discovery of the ARAS the behavioral evidence of Duffy, Freeman, and others of the "energetics" group had suggested the existence of some such brain mechanism. Moreover, learning theorists of the Hull school have in their concept of the general drive state come very close to the activation principle. Up to the present time they have employed physiological measures only sparingly and have restricted their use to the aversive aspects of drive. But with evidence that such measures may also be applied to nonaversive (appetitional) drive, it seems likely that the present rather unsatisfactory measures of drive may eventually be replaced by physiological indicants.

Activation has a number of main

characteristics that may be listed as follows: (*a*) Activation has no steering function in behavior. (*b*) It is considerably broader than emotion. (*c*) Activation is not a state that can be inferred from knowledge of antecedent conditions alone, because it is the product of an interaction between internal conditions such as hunger or thirst, and external cues. (*d*) Activation does not fit very well into the S-R formula. It is a phenomenon of slow changes, of drifts in level with a time order of minutes (even hours) not of seconds or fractions thereof. (*e*) Activation is a quantifiable dimension and the evidence indicates that physiological measures show a sufficiently high intraindividual concordance for quantifying this dimension.

It is suggested that activation is mediated chiefly through the ARAS which seems, in the main, to be an intensity system. Neurophysiological findings strongly suggest that it may be possible to achieve more precise measurement of activation through a direct recording of discharge by the ARAS into the cerebral cortex. Research on this problem is urgently needed.

The concept of activation appears to have wide application to phenomena in the field of clinical psychology. As one illustration, in this paper, activation was applied to clinical phenomena of tensional symptoms.

REFERENCES

BINDRA, D. *Motivation. A systematic reinterpretation.* New York: Ronald, 1959.

BROWN, J. S. Pleasure-seeking behavior and the drive-reduction hypothesis. *Psychol. Rev.*, 1955, **62**, 169–179.

CAMPBELL, B. A., & SHEFFIELD, F. D. Relation of random activity to food deprivation. *J. comp. physiol. Psychol.*, 1953, **46**, 320–326.

COFER, C. N. Motivation. *Annu. Rev. Psychol.*, 1959, **10**, 173–202.

COURTS, F. A. Relations between muscular tension and performance. *Psychol. Bull.*, 1942, **39**, 347–367.

DARROW, C. W. Psychological and psychophysiological significance of the electroencephalogram. *Psychol. Rev.*, 1947, **54**, 157–168.

DAVIS, R. C. Modification of the galvanic reflex by daily repetition of a stimulus. *J. exp. Psychol.*, 1934, **17**, 504–535.

DELL, P. C. Humoral effects on the brain stem reticular formations. In H. H. Jasper, L. D. Proctor, R. S. Knighton, W. C. Noshay, & R. T. Costello (Eds.), *Reticular formation of the brain.* Toronto: Little, Brown, 1958. Pp 365–379.

DUFFY, ELIZABETH. The measurement of muscular tension as a technique for the study of emotional tendencies. *Amer. J. Psychol.*, 1932, **44**, 146–162.

DUFFY, ELIZABETH. The concept of energy mobilization. *Psychol. Rev.*, 1951, **58**, 30–40.

DUFFY, ELIZABETH. The psychological significance of the concept of "arousal" or "activation." *Psychol. Rev.*, 1957, **64**, 265–275.

DUFFY, ELIZABETH, & LACEY, O. L. Adaptation in energy mobilization: changes in general level of palmar skin conductance. *J. exp. Psychol.*, 1946, **36**, 437–452.

DUSSER DE BARENNE, J. G. The labyrinthine and postural mechanisms. In C. Murchison (Ed.), *A handbook of general experimental psychology.* Worcester, Mass.: Clark Univer. Press, 1934. Pp. 204–246.

FELDMAN, S. M. Differential effect of shock as a function of intensity and cue factors in maze learning. Unpublished doctoral dissertation, McGill Univer., 1958.

FINAN, J. L. Quantitative studies of motivation. I. Strength of conditioning in rats under varying degrees of hunger. *J. comp. Psychol.*, 1940, **29**, 119–134.

FINCH, G. Hunger as a determinant of conditional and unconditional salivary response magnitude. *Amer. J. Physiol.*, 1938, **123**, 379–382.

FREEMAN, G. L. The relationship between performance level and bodily activity level. *J. exp. Psychol.*, 1940, **26**, 602–608.

FREEMAN, G. L. *The energetics of human behavior.* Ithaca, N. Y.: Cornell Univer. Press, 1948.

FUSTER, J. M. Effects of stimulation of brain stem on tachistoscopic perception. *Science*, 1958, **127**, 150.

HEBB, D. O. *The organization of behavior.* New York: Wiley, 1949.

HEBB, D. O. Drives and the C.N.S. (conceptual nervous system). *Psychol. Rev.*, 1955, **62**, 243–254.

JASPER, H. H. Electroencephalography. In W. Penfield & T. C. Erickson (Eds.), *Epilepsy and cerebral localization*. Springfield, Ill.: Charles C Thomas, 1941, 380–454.

KAPLAN, M. The effects of noxious stimulus intensity and duration during intermittent reinforcement of escape behavior. *J. comp. physiol. Psychol.*, 1952, **45**, 538–549.

KENDLER, H. H. Learning. *Annu. Rev. Psychol.*, 1959, **10**, 43–88.

KUETHE, J. L., & ERIKSEN, C. W. Personality, anxiety, and muscle tension as determinants of response stereotypy. *J. abnorm. soc. Psychol.*, 1957, **54**, 400–404.

LACEY, J. I., & LACEY, BEATRICE C. Verification and extension of the principle of autonomic response-stereotypy. *Amer. J. Psychol.*, 1958, **71**, 50–73.

LI, C. L., & JASPER, H. H. Microelectrode studies of the electrical activity of the cerebral cortex in the cat. *J. Physiol.*, 1953, **121**, 117–140.

LINDSLEY, D. B. Emotion. In S. S. Stevens (Ed.), *Handbook of experimental psychology*. New York: Wiley, 1951. Pp. 473–516.

LINDSLEY, D. B. Psychophysiology and motivation. In M. R. Jones (Ed.), *Nebraska symposium on motivation 1957*. Lincoln: Univer. Nebr. Press, 1957. Pp. 44–105.

LORENTE DE NÓ, R. Transmission of impulses through cranial motor nuclei. *J. Neurophysiol.*, 1939, **2**, 402–464.

MAGOUN, H. W. *The waking brain*. Springfield, Ill.: Charles C Thomas, 1958.

MALMO, R. B. Measurement of drive: An unsolved problem in psychology. In M. R. Jones (Ed.), *Nebraska symposium on motivation 1958*. Lincoln: Univer. Nebr. Press, 1958, 229–265.

MALMO, R. B., & SHAGASS, C. Physiologic study of symptom mechanisms in psychiatric patients under stress. *Psychosom. Med.*, 1949, **11**, 25–29.

MEYER, D. R., & NOBLE, M. E. Summation of manifest anxiety and muscular tension. *J. exp. Psychol.*, 1958, **55**, 599–602.

OLSZEWSKI, J. The cytoarchitecture of the human reticular formation. In J. F. Delafresnaye (Ed.), *Brain mechanisms and consciousness*. Springfield, Ill.: Charles C Thomas, 1954. Pp. 54–76.

ROSS, W. R. D., & DAVIS, J. F. Stable bandpass filters for electroencephalography. *IRE Canad. Convention Rec. 1958*, Paper No. 860, 202–206.

ROTHBALLER, A. B. Studies on the adrenaline-sensitive component of the reticular activating system. *EEG Clin. Neurophysiol.*, 1956, **8**, 603–621.

SAFFRAN, M., SCHALLY, A. V., & BENFEY, B. G. Stimulation of the release of corticotropin from the adenohypophysis by a neurohypophysial factor. *Endocrinology*, 1955, **57**, 439–444.

SCHNORE, M. M. Individual patterns of physiological activity as a function of task differences and degree of arousal. *J. exp. Psychol.*, 1959, **58**, 117–128.

SPENCE, K. W. Theory of emotionally based drive (D) and its relation to performance in simple learning situations. *Amer. Psychologist*, 1958, **13**, 131–141.

STENNETT, R. G. The relationship of performance level to level of arousal. *J. exp. Psychol.*, 1957, **54**, 54–61. (a)

STENNETT, R. G. The relationship of alpha amplitude to the level of palmar conductance. *EEG Clin. Neurophysiol.*, 1957, **9**, 131–138. (b)

SURWILLO, W. W. A device for recording variations in pressure of grip during tracking. *Amer. J. Psychol.*, 1955, **68**, 669–670.

SURWILLO, W. W. Psychological factors in muscle-action potentials: EMG gradients. *J. exp. Psychol.*, 1956, **52**, 263–272.

TAYLOR, JANET A. A personality scale of manifest anxiety. *J. abnorm. soc. Psychol.*, 1953, **48**, 285–290.

WOODWORTH, R. S., & SCHLOSBERG, H. *Experimental psychology*. New York: Holt, 1954.

YATES, A. J. The application of learning theory to the treatment of tics. *J. abnorm. soc. Psychol.*, 1958, **56**, 175–182.

(Received May 4, 1959)

Psychological Review
Vol. 66, No. 6, 1959

THE RELATIONAL DETERMINATION OF PERCEIVED SIZE[1]

IRVIN ROCK[2] AND SHELDON EBENHOLTZ

Graduate Faculty, New School for Social Research

One generalization that is now rather widely accepted in the field of perception is that perceptual qualities are often determined by relational rather than by absolute characteristics of the stimulus. Of the many examples that could be given to illustrate the point we will restrict ourselves to two which have certain formal similarities to the problem we wish to discuss in this paper.

The first example is concerned with the problem of neutral or achromatic color. One might ask, what is the stimulus for the experience of a given shade of gray? In the achromatic series it would seem there is no simple stimulus for neutral color corresponding to the role that wave length plays in accounting for chromatic color. Intensity of stimulation is inadequate because this changes with every change in illumination whereas the particular gray perceived remains constant. It is true that the albedo or reflectance property of an object is fixed (i.e. a white surface is one that reflects roughly 80% of the light it receives, a black, one that reflects roughly 5%), but this is a statement about the external object. The problem remains of uncovering the invariant characteristic of the stimulus that makes possible the invariant perceptual experience. Wallach's (1948) solution of this problem was that the stimulus for a particular gray is the ratio of light intensities of neighboring retinal areas. He demonstrated that a given ratio of intensities (in an otherwise completely dark room) would always yield a given shade of gray. For example (Fig. 1) a disk of Intensity 5 (arbitrary units) surrounded by a ring of Intensity 10 would appear light gray; so would a disk of Intensity 25 surrounded by a ring of Intensity 50.

The second example is concerned with the problem of perceived speed. One might speculate that the determinant of the experienced velocity of an object is the rate at which its image traverses the retina. J. F. Brown (1931) has shown, however, that velocity depends not so much on absolute rate of displacement of an image on the retina as on the rate of displacement relative to a frame of reference. He presented his observers with two equidistant fields of dots (Fig. 2) moving behind apertures in an otherwise darkened room, the linear dimensions of one field being transposed by a given multiple of the other. He found that the dots in one field would appear to be moving at approximately the same speed as those in the other, not when they moved at

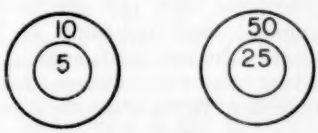

FIGURE 1.

[1] This investigation was supported by Research Grant M-2082, National Institute of Mental Health, Public Health Service. The authors wish to thank Erwin Rosen for his help in performing several of the experiments.

[2] Now at the Graduate School of Education, Yeshiva University.

FIGURE 2.

the same absolute speed, but when they traversed their respective apertures at the same relative rate. Thus, for example, when the dimensions of fields were transposed in the ratio of 2:1, equal phenomenal speed resulted only when the objective speed was also transposed by approximately 2:1. The same effect occurred, with some departure, for other ratios of transposition.

The question arises whether such relational determination may not also exist for phenomenal size. That is to say, it is possible that the stimulus correlate for an object's experienced size is not so much the absolute size of its image as it is the size of its image relative to the image size of a neighboring object (which may then serve as frame of reference). Certainly the absolute size of the image cannot fully account for phenomenal size since it changes with changes in the object's distance while the phenomenal size remains more or less constant.

To test the relational hypothesis it is necessary to show that size does depend on such stimulus relationships, i.e., that two retinal extents will give rise to approximately equal phenomenal sizes when these extents are in the same ratio to other components of their respective retinal patterns. The design called for is one in which the observer is confronted with two stimlus configurations, each consisting of two elements. In one configuration, the standard, the two elements remain fixed in size. In the other, the absolute size of one member is considerably different from that of the corresponding member of the first configuration and the second member is to be equated in size with the second member of the first configuration. The two configurations should be equidistant from the observer. In Fig. 3, for example, Rectangle B is three times larger than Rectangle A and Variable Line B is to be equated with Standard Line A. The prediction would then be that the variable line would have to be set to a length three times the size of the standard in order for the observer to see them as equal if the effect is complete or at least considerably larger than the standard if it is present but not complete. Experiments somewhat along this line have been performed in the past but not for the theoretical reasons here outlined (Künnapas, 1955; Obonai, 1954). They were performed to determine whether context would have an effect on size (in the sense of an optical illusion); only a very slight effect was obtained (of the order of about 5 to 10% difference in the size of the lines) (Künnapas, 1955). But there was a major reason why such experiments could not be expected to show a strong effect in terms of the thesis outlined above: they were done in full illumination. Under these conditions, each rectangle or frame of reference is seen within a common frame of reference (e.g. one wall of the room) as illustrated schematically in Fig. 4. In relation to the outermost common frame of reference, the two lines would only look equal when they were objectively equal and this would operate

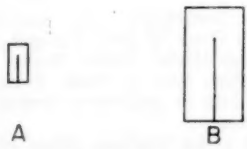

FIGURE 3.

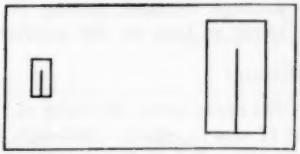

FIGURE 4.

to oppose the expected effect. Hence the experiments to be described were performed with luminous objects in total darkness just as was the case in the Wallach and Brown experiments discussed above. To eliminate a direct effect of the two configurations on one another it was also necessary to separate them in space.

Experiments 1A, B, and C

Procedure. Ten college students (five male and five female) took part in Experiment 1A. The *S* was led with eyes closed into a completely darkened room and seated exactly midway and at a distance of 5 ft. from each of two luminous rectangles having a ¼-in. luminous contour. He had to turn around 180° to look from one field to the other. The dimensions of these rectangles were 2×4 in. for the standard which appeared on the *S*'s left and 6×12 in. for the variable on his right. The smaller dimension in each rectangle served as the base. Each rectangle contained a luminous line starting at the base and running up the center.

The rectangles were produced by the application of luminous paint on black cardboard. In order to compare lengths it was necessary to have a line of variable length in one of the rectangles. This was achieved by cutting a 2 in. wide vertical slit through the center of the rectangle running from the base to the top of the luminous contour. Behind this slit was placed a strip of black cardboard upon which was painted a luminous line of desired width. This strip was then fitted with a track such that it could be moved manually, either up or down behind the luminous contour. A permanent scale of ⅛ in. intervals was placed alongside the vertical slit thus enabling the *S*'s settings to be read off directly.

In experiments where an artificial pupil was used, 8-watt ultraviolet bulbs were employed to increase the apparent luminosity of the rectangles. This was necessary because the artificial pupil severely reduced the amount of light from the rectangles reaching the eye. Where an artificial pupil was not used the ultraviolet bulbs could not be employed because the white light which they emitted would have made other objects visible under these conditions of viewing. In such experiments, therefore, the rectangles were illuminated periodically by a lamp to maintain the necessary level of brightness. In all experiments reported here wherever the luminous rectangles seemed to light up other objects in the room *S* was required to wear sunglasses. This served to keep the background completely dark.

In Part I of this experiment, the *S* was asked to equate a luminous line of variable length on his right with a standard luminous line of 3 in. length on his left when the rectangles were not yet visible. In this condition both lines were of equal width. In Part II the *S*'s task was again to equate a variable luminous line on his right with a 3 in. standard on his left. In this condition, however, the lines were presented within two luminous rectangular contours of the dimension given above, such that the rectangles were transposed in all linear dimensions in the ratio of 3:1. In order to make transposition complete, the variable line was also transposed to three times the width of the standard line. All *S*s were given one ascending and one descending trial using the method of limits. In both parts the descending trial was given with the starting position of the variable at 12 in. The *S* viewed the scene binocularly. The following instructions were read at the beginning of the experiment after a 15-min dark adaptation period.

> You may now open your eyes. If you look to your left and then to your right you will find a luminous line in each direction. I can adjust the rod on the right so that it changes its length. I want you to look to the right and tell me when it has the same length as the rod on the left. You may look back and forth as often as you like. It is important that you do not try to figure out the lengths, but try to report your spontaneous impressions. It is equally important that when you have completed your match, the lines look to you to be equal in length with respect to each other.

At the beginning of Part II, *S* was instructed as follows: "In this part of the experiment your task is exactly the same

as before" and the above instructions were repeated with the following addition to the last sentence . . . "and not necessarily with respect to the frames."

Thus the instructions were formulated in such a way as to make sure that the task was clear—that *S* could not believe he was supposed to make a relational match.

Unless otherwise specified the procedure for all subsequent experiments followed the outline reported above.

By way of supplementing these data, several other experiments were performed under only slightly different conditions. Experiment 1B was identical with 1A except that prior to the matching of the lines alone the *S*s were required to match the rectangles alone (described below as Experiment 5A). Also, the observations were made monocularly, through an artificial pupil. Fourteen *S*s were used. Experiment 1C was also identical except that it was preceded by a slightly different task involving the matching of the apparent distances of the two rectangles. An additional 10 *S*s participated in this variation. In these two experiments the last line of the instructions ("and not necessarily with respect to the frames") given in Part II of Experiment 1A was deleted but care was taken to ensure that the *S*s understood the task. If *E* had any doubt, he questioned *S*.

Results. The first three rows of Table 1 summarize the results of these experiments. The next to the last column gives the ratio of the average adjustment in Part II to the 3 in. standard.

In Part I a 3 in. standard was matched on the average with a variable length of 3.1 in. ($SD = .18$) in Experiment 1A, with 3.2 in. ($SD = .17$) in 1B and with 2.9 in. ($SD = .65$) in 1C. Whereas the identical line length was matched in Part II to an average length of 6.0 in. ($SD = 1.5$), 6.6 in. ($SD = 2.0$), and 6.8 in. ($SD = 1.7$) for Experiments 1A, B, and C.

These experiments indicate that under conditions where it is perfectly feasible to perceive lengths accurately on the basis of their actual size, the presence of the surrounding rectangles yields a very strong relational effect. The average variable s[illegible] well over twice as long as [illegible]dard.

Experiment 2

In this experiment the order of Parts I and II was reversed. Although there can be no question that the *S*s understood the instructions, the variable line in the large rectangle was now adjusted, on the average, to 7.2 in. ($SD = 1.2$) ($N = 9$). Without the rectangles present the variable line was set on the average at 3.7 in. ($SD = .66$). This setting is at a distance of approximately three times the standard error of the mean from 3.0 and thus reveals a very interesting after-effect of the adjustment in Part I. When the rectangles were present the image of the variable line was objectively larger than that of the standard when the lines appeared equal, and apparently as a result there was some tendency to continue in this direction even when the frames of reference were absent. This finding suggests that a reverse effect may have occurred in the several variations of Experiment 1, namely, an aftereffect of absolutely equal image sizes operating to oppose the transposition effect. From this point of view, one might argue that the results of Experiment 2 should be given added weight as perhaps the least biased measure of the effect under investigation (hence in all subsequent experiments Part II was given first). Using the 3 in. standard as a base, the average variable setting yields a ratio of 2.4. In other words the *S*s experience the two lines as equal when one is objectively 2.4 times larger than the other.

Experiment 3

There is one minor flaw in the experiments described above. It will be recalled that the variable line was itself transposed, such that its width was

TABLE 1

SETTINGS IN INCHES IN EXPERIMENTS 1–4

(Standard = 3 in.)[a]

Experiment	Condition	Without Rectangles		With Rectangles		Ratio Variable/ Standard	N
		M	$SD_{\bar{x}}$	M	SD		
1A	15 min. adaptation; binocular vision	3.1	.18	6.0	1.5	2.0	10
1B	Prior task; artificial pupil	3.2	.17	6.6	2.0	2.2	14
1C	Prior task; artificial pupil	2.9	.65 (N = 6)[b]	6.8	1.7	2.3	10
2	Order reversed; binocular vision	3.7	.66	7.2	1.2	2.4	9
3	Width of line varied; binocular vision			6.7	1.6	2.2	14
4A	Triangles; binocular vision	3.3	.62 (N = 10)[c]	5.2	.88	1.7	10
4B	Circles, in squares; binocular vision; standard = 1 in.	1.1	.19 (N = 8)[c]	2.0	.79	2.0	10

[a] In Experiment 4B the standard was a circle of 1 in. diameter.
[b] Only 6 of the 10 *Ss* participated in this condition.
[c] Separate *Ss* were employed for these conditions.

three times that of the standard line. This was done so that all dimensions would be transposed. But this means that if *S* were to try to match the lines when they are absolutely equal in length the difference in widths would work against such a match.

The procedure was, therefore, modified so that the variable line always preserved the proportions of the standard line. This was accomplished by presenting *S* with a series of variable lines in 1 in. steps, from 2 in. to 10 in., in ascending or descending order. Cardboard strips containing the luminous lines were inserted in a carboard slot fixed to the front surface of the variable rectangle. In this procedure the 3 in. line was therefore identical in every respect to the standard, thus meeting the above objection. The result, for the line-in-frame condition only, does not differ appreciably from that of Experiment 2. For 14 *Ss* the mean setting was 6.7 in. ($SD = 1.6$).

Experiment 4A

The foregoing experiments all employ a line figure as the critical object. To explore the effect for objects which appear phenomenally more two-dimensional, the above procedure was employed using triangles as the objects to be compared. The standard triangle in the small 2×4 in. rectangle was 3 in. high with a $1\frac{1}{2}$ in. base formed by the bottom line of the rectangle. The entire area enclosed by the triangle was luminous. The variable triangle in the large 6×12 in. rectangle was similarly constructed, but was larger such that it could be brought up from the base to any desired height. The triangle was placed behind the luminous rectangle, the inside of which was cut away. The triangle disappeared below the base of the rectangle as it was made smaller. Thus as it was moved up or down its over-all shape remained constant and similar to that of the standard. In this re-

spect the procedure is similar to that of Experiment 3. Ten *S*s were employed in this variation. The average setting was 5.2 in. ($SD = .88$). A separate group of 10 *S*s was required to match the triangles in the absence of the rectangles. The mean setting was 3.3 in. ($SD = .62$).

Experiment 4B

A similar procedure was employed using luminous outline circles in squares. The standard was a 1 in. diam. circle in the center of a 4 × 4 in. square. The variable circles appeared in a 12 × 12 in. square and were placed one by one in the center of the square in 5 steps from .5 in. to 4 in. in diameter, in ascending or descending order. The luminous circumference of the circles was approximately ⅛ in. thick. Ten *S*s partook in this variation. The mean setting was 2.0 ($SD = .79$). A separate group of 8 *S*s was given the task of equating the circles without the squares present. As in the previous experiments, this task was accomplished with considerable accuracy and consistency, the mean setting being 1.1 ($SD = .19$).

The results for 4A and 4B are somewhat lower than those for the line in otherwise comparable experiments. A possible explanation for this difference is that the triangles and circles are two-dimensional figures which, therefore, differ considerably in area when their linear dimensions differ to even a slight extent. When, for example, the heights of the two triangles are in the ratio of 2:1, their areas are in the ratio of 4:1. Figure 5 illustrates this relationship. The areal change occurs for the line figures too but seems not to exert a psychological effect—phenomenally they appear as more or less one-dimensional.

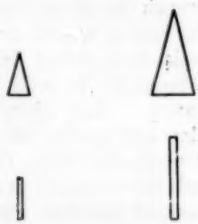

FIGURE 5.

The question arises whether the observer perceived the two rectangles to be equidistant from himself. If for any reason the small rectangle was seen as farther away—if for example it was seen as three times as far as the large rectangle—we would expect it to appear to be about the same size as the large one on the basis of the traditional explanation of size constancy, namely, taking distance into account. This is the case because a rectangle equal in size to the large one, but three times as far, would also yield an image ⅓ the size of the near one. In that event the results we obtained would not be very surprising. If the smaller rectangle were seen as equal in size to the large rectangle then naturally an observer would regard the inner forms as equal when they were both proportional in length to their corresponding rectangles. It was for this reason that we required *S* to view the rectangles binocularly in the majority of the experiments, thus hoping to ensure fairly accurate distance perception. (In Experiments 1B and 1C and other experiments we also obtained measurements under conditions of monocular vision through an artificial pupil, thereby eliminating all physiological cues to distance. Since the results were not too different from those for binocular vision they will not be discussed further.)

But it might be argued that notwithstanding the use of binocular vision, which ordinarily would allow fairly accurate distance perception, there was

a factor operating which might create the impression that the small rectangle was farther away, namely what has been called the relative size cue to distance. Two objects of similar shapes but of different sizes may create the impression of two equal sized objects at different distances. This happens in the case of the images of objects in a perspective scene and has a strong effect in drawings and photographs. It is possible that this cue outweighed the binocular cues. In order to rule out such an explanation the following two experiments were performed.

Experiment 5A

The rationale for the first experiment is as follows. If it were true that in the previous experiments the small rectangle looked farther away, and hence as large as or almost as large as the big rectangle, then when the rectangles themselves are compared this fact should be revealed. *S*s should equate the two rectangles in size when in actual fact they are quite unequal. In fact they should consider them as equal at or close to the starting size. It is important to be clear that the objection is not merely that the small rectangle appears to be farther away but that *it appears to be about equal in size to the large one because it appears farther away.*

The most obvious way of testing this deduction is to require the *S* to equate the sizes of the rectangles, varying the size of one until he considers them equal. We performed such an experiment and found that the *S*s were able to make quite accurate matches, thus suggesting that the rectangles did not at all appear equal or even nearly equal at the outset. There is, however, a flaw in this design. Suppose at the outset the smaller rectangle appears to be only about twice as far as the larger one. It will therefore appear to be about two thirds the size of the large one. (Such a partial distance effect could indeed explain our findings since only partial transposition was achieved.) The *S* would then require the variable rectangle to be changed slightly. Once this change is made, however, *S* is now confronted with a new relative-size cue to distance. On the basis of this he may again request a further size adjustment, and so on. Eventually this could also lead to an accurate match.

To get around this difficulty the experiment was revised as follows.

Procedure. After first comparing the two rectangles *S* was required to make his match from memory without further reference to the standard, which was then hidden from view. This procedure eliminates the problem of continuously changing relative-size cues to distance.

Ten college students were employed. Again the rectangles were separated from one another by 180° so that *S* had to turn his head from one to the other and the same dark room conditions prevailed. At the outset the *S* was shown the two rectangles in the size ratio of 3:1 as in the previous experiments, with the major difference that the vertical lines were not present. As in the previous experiments *S* was again at a distance of 5 ft. from each rectangle. The larger rectangle was now so constructed that its size could be varied (the top and right side formed one unit which extended in back of the bottom and left side and which could be moved diagonally upward or downward). *S* viewed the rectangles binocularly. He was told he would be asked to give his impression of the relative size of the rectangles and was given adequate opportunity to compare them with one another. Following that the standard was blocked from view and *S* proceeded to equate the variable to the size of the standard from immediate memory. He did not see the variable rectangle during the period its size was being altered.

For the decending condition the standard rectangle was 2×4 in. For the ascending condition the standard was 3×6 in. The variable rectangle was first set as larger than the standard (6×12 in.) for the

descending condition and reduced in size. For the ascending condition it was first set as smaller than the standard (1×2 in.) and increased in size. For half the *S*s the ascending condition was given first and for the other half the order was reversed. Before the second condition *S* was given an additional view of both rectangles. If there is any tendency at all to see the rectangle which is smaller as farther away, and, therefore, closer in size to the other rectangle the average setting of the variable should be considerably smaller than the standard in the ascending condition and considerably larger in the descending condition.

Results. The results shown in Table 2 were quite clear. The *S*s were able to equate the two rectangles with considerable accuracy. With the standard measuring 4 in. in height for the descending condition, the variable was set on the average at a height of 5.1; with the standard measuring 6 in., for the ascending condition, the variable was set on the average at 6.5 in. In both cases there was a slight tendency toward overestimating. The ascending average was in the direction opposite to that predicted by the hypothesis being tested. Thus it must be concluded that in the earlier experiments either the smaller rectangle did not appear to be appreciably farther away, or if it did, its apparent distance did not affect its size to any substantial extent.

TABLE 2

MEAN SETTINGS OF HEIGHT OF RECTANGLES IN EXPERIMENT 5A

	Total Ascending (6 in. Standard)	Total Descending (4 in. Standard)	Ascending of *S*s given ascending first	Descending of *S*s given descending first
M	6.5	5.1	6.6	4.4
SD	.36	.63	.82	.40
N	10	10	5	5

Experiment 5B

As a matter of fact, in a further experiment it was possible to test directly the question of a distance effect. Again the observer was confronted at the outset with the two different sized rectangles as in Experiments 1–4, each at 5 ft. The larger one was now set on a track and the observer was required to indicate when it appeared equidistant with the smaller one (here using binocular vision). As in Experiment 5A *S* was not allowed to view the two rectangles together once he began making his match. Two matches were made by each *S* but they were both of necessity descending trials, i.e. the large rectangle was moved away from *S* in both cases. The average setting was at 5.6 ft. for 7 *S*s excluding one who saw no difference and two who saw the larger rectangle as farther away. On the average, then, the larger rectangle appeared only slightly nearer than the smaller one.[3]

Returning to the main findings, an important question arises at this point. The results show that to a very considerable extent size is determined relationally. Yet the average setting of the variable stimulus falls short of the value required for complete proportionality. What factor (or factors) opposes relational determination?

It is always possible to argue that we did not achieve complete darkness and that, therefore, to some extent a common frame of reference was present. If true it would be expected to oppose a proportionality outcome as noted earlier. We did perform an experiment similar to those reported above using lines, but with the room lights on. The ratio than dropped to

[3] The absence of a marked effect of the relative-size cue in this experiment is probably due to the fact that the two rectangles are 180° apart with respect to the *S*. Ordinarily when this cue is effective the objects are more or less adjacent to one another.

1.3:1, so there is no question that a common frame would have a deleterious effect. But the fact is that great pains were taken to ensure total darkness in these experiments and a careful inspection by two observers failed to reveal any visible objects other than the luminous objects proper.

More probably, relational determination is not complete here because the size of the inner objects *relative to one another* is at least to some extent still perceptible in spite of their being surrounded by their respective rectangles and separated from one another spatially. The results of the figures-alone matches show clearly that in the absence of the rectangle the figures can be equated to one another with considerable accuracy. This being the case, the magnitude of the departure from objective equality with the rectangles present must be considered as an impressive example of what Duncker (1929) has described as separation of system, albeit a separation which is not complete. A relational match also implies that *S* must ignore the logical contradiction that objects proportional to rectangles which themselves are perceived as quite unequal cannot be equal to one another.

If it is correct that the size of the inner objects relative to one another is to some extent still perceptible, it would follow that for transposition ratios greater than 3:1 there would be a falling off of the relational effect. As transposition ratios are increased, the relative sizes of the enclosed lines must diverge more and more from absolute equality in order to yield relational matches. For example a 5:1 ratio would require one object to be set to a size five times the size of the other. Although a strong tendency in this direction might exist it would be opposed by the very great absolute size difference required. Conversely it follows that for a ratio less than 3:1 the relational effect would increase because the observer would only have to tolerate a smaller absolute difference.

TABLE 3

	Transposition Ratio			
	2:1	3:1	5:1	8:1
N	10	14	10	10
M	6.4	6.7	6.6	6.2
SD	.30	1.6	1.0	1.0
Standard	4	3	3	1.8
Ratio	1.6	2.2	2.2	3.4
% Complete	80%	73%	44%	42.5%

Experiment 6A, B, and C

The procedure of Experiment 3 was followed in all respects except for the difference in the sizes of the two rectangles. In Experiment 6A the smaller rectangle was 3 × 6 in. and the larger one was 6 × 12 in., thus yielding a ratio of 2:1. In Experiment 6B the smaller rectangle was 2 × 4 in. and the larger one was 10 × 20 in., thus yielding a ratio of 5:1. In Experiment 6C the smaller rectangle was 1.2 ×2.4 in. and the larger one was 9.6 × 19.2 in., thus yielding a ratio of 8:1.

The results together with those of Experiments 3 for the 3:1 ratio are shown in Table 3.

The results show that as the transposition of dimensions increases, the ratio of the variable setting to the size of the standard increases. For the 8:1 transposition the variable line is set on the average at 3.4 times the size of the standard. This is a very impressive relational effect. Nevertheless it is true that in comparison with what would be complete transposition there is a falling off of the effect.[4] Hence

[4] In the transposition of velocity, Brown (1931) found a less than complete effect for fields transposed in the ratio of 2:1 and a falling off of the effect with fields trans-

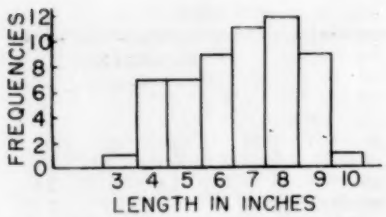

FIG. 6. Length in inches of variable setting of line matched to the 3 in. standard.

these results support the view that it is the impression of the size of the inner objects relative to one another which opposes the relational effect.[5] Separation of system is not complete.

There were, however, large individual differences. For example in Experiments 1A, B, C, 2, and 3 close to half the sample tested gave complete or nearly complete proportional matches and others gave matches closer to objective equality. Figure 6 shows the distribution of matches for all *S*s combined in these five experiments. These differences no doubt reflect the way in which *S*s resolved the conflict between the relational effect and the impression of the size of the inner objects to one another.[6] Individual consistency was, however, very high. The correlation between ascending and descending trials for the *S*s in these five experiments was .93.

One of the major implications of the relational basis of achromatic color and speed perception is that it offers an explanation of achromatic color and speed constancy (Wallach: 1939, 1948). When in daily life the illumination changes it almost always affects all neighboring regions equally. Since the *ratio* of reflectances is not changed thereby, there is no reason to expect the experienced color to change if it is true that achromatic color depends upon the ratio of reflectances. Similarly when a moving object is viewed at varying distances its rate of displacement relative to some frame of reference (a car passing along a road in front of a row of trees for example) does not change. If phenomenal speed depends on rate of relative displacement as Brown has shown—and a change in distance only brings about a transposition on the retina of all the dimensions of the scene—there is no reason to expect any change to occur with variations in distance.

Now that we have been able to demonstrate a rather strong relational determination of phenomenal size it is plausible, therefore, to inquire whether this can account for size constancy. When we see an object next to some other object of a different size, one which we might say serves as a frame

posed in greater ratios. However in absolute terms the transposition of velocity is more nearly complete than is the transposition of size.

There are several possible ways of explaining the superiority of transposition of velocity. Duncker's (1929) work leads to the conclusion that movement is itself relationally determined and movement is the quality of which speed (rate of movement) is the quantitative aspect. But extensity—of which size is the quantitative aspect—is not relationally determined. Furthermore the perceived discrepancy in the *size* of the two rectangles is bound to be more disturbing to the relational determination of the *size* of figures than to the *speed* of figures—i.e., size can be compared with size more directly than with speed. Finally it might be easier to compare the sizes of figures without rectangles present than the speed of objects, and this being the case the absolute comparison might also obtrude itself more in the case of size than of speed.

[5] The poorer results with the two-dimensional figures also support this view because the absolute size-differences were more obtrusive.

[6] Females showed a slight tendency toward higher settings than males, namely 6.8 in. vs. 6.4 in. in the same five experiments cited above. The difference, however, falls short of significance.

of reference, the ratio of the two sizes to one another and, therefore, of their respective image sizes would remain constant with changes of distance. To the extent that phenomenal size is dependent not upon the absolute size of the image but on its size relative to another object, size constancy would follow as a matter of course, at least in many situations in daily life.

This explanation is quite different in its implications from the traditional one that distance is somehow "taken into account." The essence of this explanation as with those of Wallach is in the specification of certain invariant properties of the stimulus which can be correlated with the perceptual experience in question (size, color, speed) even when the vitiating factor (change of distance, change of illumination) operates. Thus it would no longer be correct to say that the perceptual experience in the case of a given constancy is not in perfect correspondence with the stimulus. It is only not in correspondence with that aspect of the stimulus which previously was thought to be the relevant one (size of the image, intensity of the image of the object alone, absolute rate of retinal displacement).

Another implication of the relational explanation is that it would seem to make the assumption of learning gratuitous. If we can point to an attribute or dimension of the stimulus which always corresponds with a given experience then it is no more necessary to argue that these constancies are learned than it is to argue that the dependency of hue on wave length is learned. The constancies discussed above follow as a matter of course from the very stimulus conditions which have been discovered to underlie the experienced attributes in question (size, achromatic color, speed.[7] A further implication of this type of explanation is that the constancies are truly sensory in character and are not merely intellectual elaborations of underlying sensations.

It is perhaps worth noting in passing that these solutions are compatible not only with Gestalt theory but also with other points of view such as that of Gibson (1950). Since they represent instances where relational stimulation underlies perceptual experience it is implied that the neural correlate involves central interaction processes. However one may merely prefer to say, as would Gibson, that these are precisely the kind of higher order stimulus attributes we should expect to find as the correlates of particular perceptual experiences.

But there are a number of considerations which suggest that in the case of size constancy the relational explanation is not sufficient. Unlike the case of achromatic color—where the determination by ratio is all but perfect—and the case of speed—where the transposition effect is sufficiently large to account for speed constancy—the transposition effect for size is far from complete as noted above. Yet size constancy is more or less perfect within a considerable range of distances. Certainly in many situations in daily life involving objects at distances from the observer in the ratio of say, 3:1, size constancy is complete. (At these distances the images of the two patterns would be transposed in the ratio of 3:1 and for this ratio the relational effect is not complete.) At least we can say that in experiments in which the observer is to equate sizes of ob-

[7] Nevertheless, in spite of this reasoning it is still possible that the judgment of size on the basis of stimulus relationships such as we have shown to exist may in point of fact be the result of learning.

jects at these relative distances, the results will show approximately perfect constancy unless very great absolute distances are involved. It may be argued, however, that in this situation the observer is oriented to give the "correct" answer more than he is to give a careful phenomenological description of what he *sees*. Hence to some extent he may *perceive* the distant object as at least somewhat smaller than the near one but *judge* it as equal —and to that extent Helmholtz would be correct.[8] This same objective attitude of the observer would in our situation, however, operate to reduce the relational effect.

There is no doubt some truth in this reasoning, but its importance should not be exaggerated, because when one encourages the observer to try to match in terms of visual impression instead of judgment of true size, the results are not very much different (except perhaps at great distances where there does appear to be a falling off of constancy). A more probable explanation of the fact that constancy is better than could be predicted on the basis of our results is that relational determination is not the only factor operating. It would seem necessary to admit the truth of the traditional explanation as at least one of the factors at work in size constancy for the following reason. In a completely dark room phenomenal size is not determined exclusively by visual angle relations even under conditions where an explanation in terms of a relational effect would be ruled out. A luminous object will not appear the size that its visual angle would demand when it is viewed at varying distances, e.g., it will not appear half as small at twice the distance. In short, distance is taken into account with the result that there is a tendency in the direction of constancy, and as the degree of constancy increases distance perception is better.[9]

Thus it seems possible that under ordinary circumstances both the relational factor and the taking into account of distance operate together to yield perfect or nearly perfect constancy. In the following experiment an attempt was made to examine this possibility.

Experiment 7A

Procedure. In Part I, *S* was to match a variable luminous line, at 2 ft. with a standard 3 in. line at 10 ft. in a darkened room, the two being separated by an angle of 90°. The two lines were here of equal width (.1 in.). Hence the method of Experiments 1 and 2 was used in which the length of the variable line was continuously changed. *S* was only allowed to use one eye on the assumption that this would considerably reduce the accuracy of distance perception but would not completely eliminate all cues to distance (an artificial pupil was *not* employed). Hence it was expected that the match would not be based exclusively on equality of visual angle but in-

[8] This attitude of the observer explains the apparently enigmatical fact, reported with increasing frequency during the last decade, of overconstancy, particularly at great distances. It is quite clear from the standpoint of phenomenological description that the very distant object *looks tiny*, but the experimental finding is in the opposite direction. This is what we would predict if the observer were *judging*, and in doing so, compensating for what he thinks the effect of distance is on apparent size.

[9] Recently a few psychologists have voiced their skepticism of this principle because of the fact that phenomenal size does not correlate with phenomenal distance in experiments where the observer is required to judge both (e.g. Gruber, 1954). But this conclusion is based on a premise which may be faulty. It is more likely "registered distance" as given by various cues, not "phenomenal distance," that enters into the interaction. That is, it is not essential for the distance cues to be translatable into a corresponding awareness of distance in order for them to interact with image size in yielding size constancy.

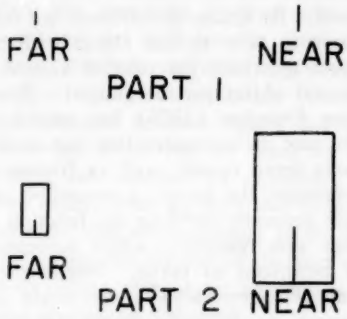

FIG. 7. The retinal situation based on the average matches in Parts I and II of Experiment 7A.

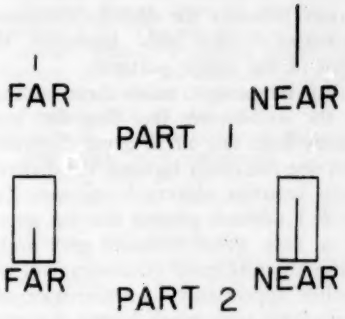

FIG. 8. The rentinal situation based on the average matches in Parts I and II of Experiment 7B.

stead would show some tendency in the direction of constancy.

In Part II, the same task was repeated except now each line was .5 in. wide and was surrounded by a luminous rectangle 6 × 12 in.

Results. In Part I, the average setting for 16 *S*s was 1.2 in. (*SD* = .34). A match based exclusively on visual angle would have required a line .6 in. in length (3 in./5 since the near line is at ⅕ the distance) whereas perfect constancy would have required a line 3 in. in length. Thus there is some slight tendency in the direction of constancy.

In Part II, the average setting was 2.7 in. (*SD* = .75) which means that here constancy was virtually complete. Since the only change was the addition of the surrounding rectangles (which of course as images were transposed in the ratio of 5:1) this increase is to be attributed to the relational effect (see Fig. 7). In this particular situation, therefore, involving only rather poor cues to distance, relational determination makes a much greater contribution to constancy than does the taking into account of distance. But the main point of this experiment is that we have shown that the relational factor does influence constancy and that when the two factors cooperate, constancy approaches completeness.

Experiment 7B

It is , of course, possible that the relational factor may interact with the distance factor in a negative sense, i.e., by yielding a nonveridical, illusory outcome. This might occur wherever the two frames of reference are objectively unequal in size. We were able to demonstrate this effect in an experiment where the lines alone at distances from the *S* in the ratio of 5:1 were first matched in the dark with *binocular* vision. This yielded perfect constancy on the average for 10 *S*s. When, however, the distant line was surrounded by a rectangle 5 times as large as the near rectangle (thus yielding equal sized images of the two rectangles), it brought the average length of its line up to 2.7 times the length of the near line (see Fig. 8).

Another problem to be considered, bearing on the applicability of our findings to size constancy, is the magnitude of the relational effect for transposition ratios other than 3:1. On the basis of the results reported above we would have to predict a falling off of size constancy as the ratio of dis-

tances between the objects compared increases — since this increases the ratio of the image patterns.

It is tempting to relate these findings to the well-known fact that size constancy does fall off at great distances. But one can easily increase the distance ratio between objects from, say, 2:1 to 8:1 without placing the far object at a very great distance and under these circumstances constancy will not decline appreciably. Furthermore one can create very much higher distance-ratios by placing the near object very close rather than the far object very far, and we have been unable to show a falling off of constancy in such a situation. In fact Jenkins (1957) recently performed such an experiment and obtained the opposite effect. However it is difficult even to demonstrate the falling off of constancy at great distances because the observer is governed by an objective attitude and not a phenomenological one in the constancy situation. This problem therefore remains open, but at the moment the facts concerning increased distance ratios do not support the relational interpretation. Perhaps the possibility of taking distance into account serves to offset the predicted decline in constancy based on the relational factor.

There are two other difficulties for the relational explanation of size constancy. One is the fact that the relational effect is smaller for figures which appear two dimensional than for the phenomenally more or less one dimensional line figures. Yet most objects in daily life present us with a two dimensional surface.

The second is that the objects we view are not necessarily always seen in front of or adjacent to a large object which can serve as a frame of reference. And even when they are, there remains the question of the constancy of size of the frame of reference itself. By frame of reference we only mean to refer to that object with respect to which the relative size of a second object can be gauged. However Duncker (1929) has shown in the case of movement that not all objects serve equally well as frames of reference, the larger, surrounding object generally fulfilling the function of that with respect to which movement is perceived to occur. Furthermore only for surrounded objects could one expect to have a strong tendency toward separation of system. We were, in fact, able to show in an experiment that when the *inner* objects were transposed in a given ratio their effect on the size of the outer rectangles was nil. This being the case the relational effect does not contribute very much to an explanation of the constancy of objects which play this role in any particular situation.

SUMMARY

By way of summary, we have shown that to a very considerable extent phenomenal size is relationally determined. The effect is greater for line figures than for those which appear as two dimensional. As the transposition of dimensions increases the ratio of the size of the variable to that of the standard increases, but, nevertheless, there is a falling off of the effect from the standpoint of complete relational determination.

We have shown that the relational effect is not an artifact based on a tendency to see the smaller frame of reference as farther away and, hence, as equal in size to the nearer one.

It would seem that the fact that the effect is not complete is due to the tendency of the observer to be influenced by the absolute sizes of the enclosed figures with respect to one another, at least to some extent.

We have considered the possibility that the relational determination of perceived size explains size constancy. Since, however, size constancy is often perfect but the transposition effect is not complete we have argued that size constancy must also be based on the taking into account of distance. In two experiments we were able to demonstrate that the relational effect *and* the taking into account of distance can both play a role in the perception of size at varying distances.

REFERENCES

BROWN, J. F. The visual perception of velocity. *Psychol. Forsch.*, 1931, **14**, 199–232.

DUNCKER, K. Über induzierte Bewegung. *Psychol. Forsch.*, 1929, **12**, 180–259.

GIBSON, J. J. *The perception of the visual world.* Boston: Houghton Mifflin, 1950.

GRUBER, H. E. The relation of perceived size to perceived distance. *Amer. J. Psychol.*, 1954, **67**, 411–426.

JENKIN, N. Effects of varied distance on short-range size judgments. *J. exp. Psychol.*, 1957, **54**, 327–331.

KÜNNAPAS, T. M. Influence of frame size on apparent length of a line. *J. exp. Psychol.*, 1955, **50**, 168–170.

OBONAI, T. Induction effects in estimates of extent. *J. exp. Psychol.*, 1954, **47**, 57–60.

WALLACH, H. On constancy of visual speed. *Psychol. Rev.*, 1939, **46**, 541–552.

WALLACH, H. Brightness constancy and the nature of achromatic colors. *J. exp. Psychol.*, 1948, **38**, 310–324.

(Received June 9, 1959)

LOOKING FOR A BOOK PUBLISHER?

It is no secret that publication is regarded as the foundation stone of a scholar's career. In order to gain recognition, prestige, and advancement, the scholar must seek publication.

Exposition Press offers scholars a complete publishing service, under our special academic imprint, Exposition-University Books. Though the proportion of scholarly publications in the lists of the trade houses is shrinking, due to economic pressures, several of our recent titles in the academic fields have gained popular reviews, professional recognition, and steady sales. These books are the result of a publishing plan that is bringing a steady flow of scholarly writers to Exposition Press. They reflect the high editorial standards and quality of design and production which have won the respect of libraries, schools, booksellers and critics.

FREE! The behind-the-scenes story of book publishing revealed in two fact-filled, illustrated brochures, containing a detailed description of our 40%-royalty subsidy plan including a breakdown of contract terms and typical costs (in print for the first time). Copies are available on request. Your inquiries and manuscripts are invited. An editorial appraisal will be furnished promptly without obligation.

Please write to Jerrold Paine, Editorial Department.

EXPOSITION PRESS, *Main Office, 386 Park Ave. S., N.Y. 16, N.Y.*

THE BRITISH JOURNAL OF PSYCHOLOGY

Edited by BORIS SEMEONOFF

Vol. 50 Part 3 August, 1959 20s. net

IAN M. L. HUNTER. The solving of five-letter anagram problems.

E. F. MACKWORTH and J. F. MACKWORTH. Remembering advance cues during searching.

R. H. DAY. Hue differences and brightness differences as determinants of figural after-effects.

GEORGE A. TALLAND. Inter-sensory perceptual set.

RAYMOND B. CATTELL and RICHARD W. COAN. Objective-test assessment of the primary personality dimensions in middle childhood.

MARGARET DONALDSON. Positive and negative information in matching problems.

DALBIR BINDRA. An interpretation of the 'displacement' phenomenon.

JOHN LEGGETT. The paired use of projective stimuli.

VERNON HAMILTON. Theories of anxiety and hysteria—a rejoinder to Hans Eysenck.

CORRIGENDUM.

PUBLICATIONS RECENTLY RECEIVED.

The subscription price per volume, payable in advance, is 60s. net (post free).

Subscriptions may be sent to any bookseller or to the

CAMBRIDGE UNIVERSITY PRESS

Bentley House, Euston Road, London, N. W. 1